A STUDY OFPERIPHEAL NERVOUS SYSTEM ALTERATIONS IN HYPOTHYROID PATIENTS

Dissertation submitted for

M.D. (Branch – V Physiology)

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

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PSG Institute of Medical Sciences & Research
Institutional Human Ethics Committee

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March 15, 2013

To
Dr M Jeyabanu
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Ref.: Your study entitled 'To study the peripheral nervous system alterations in hypothyroidism'

Ref.2: Your letter dated 01.03.2013

Sub.: Ethics Committee Approval

We have received your letter dated 01.03.2013 requesting for change in the title and aim (excluding the visually evoked potential from the study). We have also received copy of the modified Informed Consent Forms vide the above letter.

The Institutional Human Ethics Committee, PSG IMS & R, Coimbatore -4, has reviewed and discussed the above documents on 8th February, 2013 in its expedited review meeting held at College Council Room, PSG IMS&R, between 3.00 pm and 4.00 pm. After due consideration, the Committee has decided to approve the above documents.

After due consideration, the Committee has decided to approve the above study proposal.

The members who attended the meeting, at which your study proposal was discussed, are listed below:

Name	Qualification	Responsibility in IHEC	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
Dr P Sathyan	DO, DNB	Clinician, Chairperson	Male	No	Yes
Dr S Bhuvaneshwari	M.D	Clinical Pharmacologist Member – Secretary	Female	Yes	Yes
Dr Sudha Ramalingam	M.D	Epidemiologist Alt. Member – Secretary	Female	Yes	Yes
Dr Y S Sivan	Ph D	Member – Social Scientist	Male	Yes	Yes

The approval is valid for one year.

We request you to intimate the date of initiation of the study to IHEC, PSG IMS&R and also, after completion of the project, please submit completion report to IHEC.

PSG Institute of Medical Sciences & Research
Institutional Human Ethics Committee

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This Ethics Committee is organized and operates according to Good Clinical Practice and Schedule Y requirements.

Non-adherence to the Standard Operating Procedures (SOP) of the Institutional Human Ethics Committee (IHEC) and national and international ethical guidelines shall result in withdrawal of approval (suspension or termination of the study). SOP will be revised from time to time and revisions are applicable prospectively to ongoing studies approved prior to such revisions.

Kindly note this approval is subject to ratification in the full board review meeting scheduled on 28.03.2013.

Yours truly,

Dr S Bhuvaneshwari
Member - Secretary
Institutional Human Ethics Committee

SECRETARY
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CERTIFICATE

This is to certify that the dissertation work entitled “A study ofperipheral nervous system alterations in hypothyroid patients” submitted by Dr. M. Jeyabanu, is the work done by her during the period of study of herpost graduation in Physiology from June 2012 to March 2015 in our institution. This work is done under the guidance of Dr.Nagashree, HOD, Professor, Department of Physiology, PSG IMS & R.

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DECLARATION

I hereby declare that this dissertation entitled “A Study of Peripheral nervous system alterations in hypothyroid patients”was prepared by me under the guidance and supervision of Dr.Nagashree, HOD, Professor, Department of Physiology, PSG IMS&R.

This dissertation is submitted to Tamil Nadu Dr. MGR Medical University in fulfillment of the university regulations for the award of MD Degree in Physiology.

M.JEYABANU

Post graduate student

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ABSTRACT

TITLE

A study of peripheral nervous system alterations in hypothyroidism.

INTRODUCTION

Hypothyroidism is an endocrine disorder of deficient thyroid hormone levels in the circulation. Thyroid hormones are essential for the normal functioning of the brain and nervous system. One of the manifestations of the hypothyroidism is the peripheral neuropathy.

METHODS

This cross sectional study includes 30 hypothyroid patients and 30 normal subjects between the age group of 20 to 60 years. The nerve conduction study was done using Recorders Medicare System (RMS) EMG EPM2K version-1.

Three parameters (latency, amplitude and nerve conduction velocity) of motor and sensory component of three nerves (Median nerve, Ulnar nerve and Peroneal nerve) were compared between cases (Hypothyroidism) and controls (non hypothyroid). Statistical analysis was done by unpaired ‘t’ test and ANOVA with SPSS software for various analysis.

RESULTS

The nerve conduction velocity is reduced in right median, right and left ulnar and left common peroneal nerves. The latency is prolonged in the right and left ulnar nerve as well as in the peroneal (both right and left) nerve. The amplitude of the nerve conduction action potential of the all the nerves is not significantly reduced. The sensory nerve conduction velocity of the sural nerve is reduced in our present study. There is no correlation between the nerve conduction abnormalities and the age and duration of the disease in hypothyroid patients.

CONCLUSION

The prevalence of neuropathy in hypothyroid patients attending the PSG Institute of Medical Science and Research, Coimbatore is 56.66 %. In this study 36.66% hypothyroid patients (11) were found to be with carpal tunnel syndrome. The physiological parameters (age and duration of the disease) were not correlated with nerve conduction values. The median nerve was the most affected nerve in the upper limb and the sural nerve was the commonly affected nerve in the lower limb.

KEY WORDS

Hypothyrodism, Nerve conduction study

INTRODUCTION

The thyroid gland is the one of the largest endocrine glands. The Greek word ‘thyreos’ means ‘shield’ and ‘eidos’ means ‘form’. So it yields its name as it is shield shape in nature. It consists of two lobes connected by an isthmus and located anterior to the trachea between the cricoids cartilage and the suprasternal notch. Normally the thyroid gland is 12 to 20 g in size, soft and highly vascular. The thyroid gland develops from the floor of the primitive pharynx during the third week of gestation.

The thyroid secretes two important hormones named thyroxine (T_4) and triiodothyronine (T_3). These hormones act through the thyroid hormone receptors α and ß by which it plays important physiological role on most of the organs and tissues of the body. It has a critical role in cell differentiation during fetal development and helps to maintain thermogenic and metabolic homeostasis in adults are well known for a long period. An overproduction result in hyperthyroidism or deficient hormone secretion leads to hypothyroidism.

REGULATION OF T_3 AND T_4:

The pituitary gland is situated at the base of the brain in the sellaturcica of the sphenoid bone. The anterior pituitary gland secretes the most important hormone (TSH) Thyroid stimulating hormones along with other hormones. TSH controls the secretion of thyroid hormones (T_3 and T_4).The normal plasma concentration of TSH is 0.3 to 5 μ U/ml. Its secretion is mainly controlled by two factors:

1. The major stimulant for the secretion of TSH is TRH from hypothalamus.

2. Somatostatin another substance secreted from the hypothalamus inhibits the TSH secretion

The negative feedback mechanism by the thyroid hormones T_3 and T_4 inhibit the secretion of TRH. The TSH secretion is inhibited by dopamine, another hormone secreted from the hypothalamus. Cortisol and growth hormone also inhibit the TSH secretion.[2]

HYPOTHYROIDISM

It is a disorder in which the thyroid gland is unable to synthesize and secrete sufficient amounts of thyroid hormone to meet the requirement of the brain and peripheral tissues.

1. Primary hypothyroidism refers to thyroid failures that result from disease of the thyroid gland itself. This condition accounts for over 99% of all the cases of hypothyroidism. [3]

2. Central hypothyroidism/ Secondary hypothyroidism is the term for the thyroid failure caused by pituitary or hypothalamic disorders that result in deficient pituitary production of thyroid-stimulating hormone (TSH).[3]

3. Overt hypothyroidism describes moderate to severe thyroid failure resulting in high serum TSH levels (TSH >10 μIU/L) associated with low serum concentrations of total thyroxine (T_4) or free T_4.

4. Subclinical hypothyroidism[4] defined biochemically as association of raised serum TSH (above normal range of 0.5 to 5 mIU/L) with normal circulating concentrations of free T_3 and T_4.

The incidence of hypothyroidism is estimated to be 4 to 5 / 1000 population per year for women and 0.6 to 0.9 /1000 population per year for men. The prevalence of overt hypothyroidism is approximately 1% to 2% in women and 0.1% in men.[3] The cause for the hypothyroidism could be autoimmune disorder, thyroid surgery, radiation therapy pituitary disorder or iodine deficiency. Among all the most common cause is iodine deficiency.[3]

SIGNS AND SYMPTOMS OF HYPOTHYROIDISM [5, 6]

- Easy fatigability
- Increased sensitivity to cold
- Hypotonia
- Ataxia, tremor , dysmetria
- Polyneuropathy
- Entrapment neuropathy
- Slowing voluntary movement
- Myopathic weakness

- Dry skin
- Muscle pain, tenderness, stiffness
- Muscle weakness
- Pain and stiffness of joints
- Slowed heart rate
- Depression
- Impaired memory

PERIPHERAL NEUROPATHY:

One of the manifestations of the hypothyroidism is the peripheral neuropathy. The development of this neuropathy is insidious in onset, which will take a longperiod of time for clinical manifestations.

Peripheral neuropathy is divided in to three types[7].

1. Mononeuropathy,
2. Mononeuropathy multiplex/mononeuropathy of multiple single nerves &
3. Polyneuropathy

Injury to a single nerve produces a condition known as mononeuropathy. Long nerves in the upper limb or forearm or thigh or shin

region are the common sites of involvement of mononeuropathy. The nerve compression is the single most common mechanism of injury in mononeuropathies, but it may result from vasculitis or local ischemia.

Polyneuropathy is the involvement of multiple nerves. In the middle age people the prevalence of polyneuropathy is approximately 2.4% but in aged population of above 55 years of age, the incidence increases to 8 %. [7]

Features commonly observed in sensory and motor polyneuropathy:

SYMPTOMS: [7]

Early features:

1. Distal numbness and tingling
2. Distal neuropathic pain
3. Gait imbalance

Features in the later stage of the disease:

1. Progression of distal numbness and tingling to proximal body parts
2. Prominent neuropathy pain.
3. Worsening gait
4. Frequent falling

SIGNS:

Early signs:

1. Distal sensory loss to cold, pinprick and or vibration

2. Decreased are lost ankle reflex

3. Romberg sign

4. Impaired tandem walking

Features in the later stage of the disease:

1. Worsening of distal sensory loss to cold, pinprick and or vibration / joint position sense areflexia at ankle and knee.

The neuropathy when applied to the peripheral nerves system refers to disease at the level of the anterior horn cell (or), more commonly at the dorsal root ganglion. In a dorsal root gangliopathy, the sensory loss can be profound and often does not confirm to the socking and glove pattern of sensory loss, normally observed in a distal dying back neuropathy.

EFFECTS ON NERVOUS SYSTEM

The role of thyroid hormone is vital in the timing and pace of development of central nervous system of our body during intrauterine life and in the early infancy. If there is a deficiency of thyroid hormone, it will greatly influence the growth of the cerebral and cerebellar cortex, proliferation of axons and branching of dendrites, myelinization and cell migration. So thyroid hormone deficiency should be recognized as early as possible in the post natal life and prompt treatment is inevitable to avoid the irreversible brain damage.

Thyroid hormone deficiency during the critical period of neural differentiation produces permanent and severe alterations in the morphology and function of the nervous system leading to cretinism. Cognitive neurological symptoms are common in myxoedema, in particular a general slowing of cognitive functions with memory impairment and apathy[8].

EFFECTS ON PERIPHERAL NERVOUS SYSTEM

In hypothyroidism mononeuropathy and polyneuropathy are reported in previous studies. Mononeuropathy is the mucinous deposits which compresses the nerve and causes nerve damage which can be easily demonstrated by a

nerve conduction velocity studies. The involvement of primarily the myelin sheath has been revealed by some studies, [9,10] but some other studies show the primary axonal damage by the morphologicalevaluation of the nerve fibers [11,12].

Hypothyroidism affects all peripheral nerves, but more commonly affected nerve is the median nerve which results in carpal tunnel syndrome. The sensory nerve conduction deficit is more during the early stage of neuropathy, the clinical symptoms includes pain, cramps, parasthesia of fingers and limbs. It has been proved since earlier that the thyroid hormone increases the speed and amplitude of peripheral nerve reflexes.

A light and electron microscopic study of peripheral nerve and muscle done on myxoedematous polyneuropathy patients, shows segmental demyelination of the sural nerve with mucinous deposits [10].

Myxoedema is associated with neuropathy and myopathy along with the neuropsychiatric manifestations like forgetfulness, blurred vision, psychosis, convulsions and coma. 79% of the hypothyroid patient's complaints

neuromuscular problems; 38% presented with clinical weakness. Among them 42% had sensorimotor axonal neuropathy and carpal tunnel syndrome were reported in 29% of the patients included in a study done at Netherland[13].

EFFECTS ON MUSCULOSKELETAL SYSTEM:

Since 60th century the relationship between hypothyroidism and muscle disease is well known [14, 15]. Muscle pain, stiffness, arthralgia, synovial thickening and effusion, myopathy, cramps and stiffness are common features reported by the hypothyroid patients[16] other symptoms includes pseudomyotonia with delayed relaxation of the muscle and prolonged tendon reflex relaxation time.

ELECTRODIAGNOSTIC STUDIES:

1. NCS - Nerve conduction study

2. EMG – Electromyogram are the two types of electro diagnostic studies. These studies determine whether the neuropathy is due to damage to the axon (Axonal Neuropathy) or myelin (Demyelinating neuropathy) or both.

NERVE CONDUCTION STUDY:

The nerve conduction velocity test is the other name for the nerve conduction study. It is the study that measures the speed of electrical impulse conducted through the nerve. This test is valuable to determine if there is any damage to the nerve or its abnormal conduction velocity. It assesses the amplitude, latency and conduction velocity of an electrical impulse conducted over the nerve to be tested. If there is an axonal loss, NCS will show lower amplitudes and prolonged latency. Where as in case of demyelination slow conduction velocities will be the finding. [17]

The surface electrodes used in the nerve conduction study is called as patches, which is similar to those used for recording of the Electrocardiogram. The patches are placed on the skin that lies over the nerve we are going to study. These surface electrodes will give off a very minimal electrical impulse that will stimulate the nerve. That will result in electrical activity of the nerve and can be recorded by the other electrodes.

The speed of the nerve impulse is determined by the recording the distance between the stimulating electrode and the receiving electrodes. We have to repeat the same procedure to each nerve being tested. The onset latency, duration of the sensory nerve action potential, amplitude and nerve conduction velocity is the parameters we are recording by nerve conduction study.

ELECTROMYOGRAPHY (EMG)

It is another study used to differentiate the muscle and nerve injury. Electromyography refers to recording of action potentials of muscle fibers firing singly or in groups near the needle electrode in a muscle. In needle EMG, following three types of activities are studied:

1. Insertional activity

2. Spontaneous activity

3. Voluntary activity.

A fine needle is inserted into the muscle under the study, to compare the amount of the electrical activity during the rest and during the contraction of the muscle. Both procedures help to detect the presence, location, and extent of diseases that damage the nerves and muscles.[16]

Eslamian F et al[18] evaluated the signs and symptoms of neuromuscular dysfunction in primary hypothyroid patients. They found that the patients with hypothyroidism had the clinical features of mononeuropathy, proximal muscle weakness and sensorimotor polyneuropathy.

MarciaW et al[19] studied 16 patients with primary hypothyroidism. The ENMG and NCS findings are:

1. Motor nerve latency prolonged in median nerve and peroneal nerve
2. ulnar and sural nerve conduction velocity reduced

The neurological complications in hypothyroidism are well proved findings. Another study done during 1980 shows the prevalence of neuropathy in hypothyroid patients ranges from 10% to 70 %. [20]

AIM AND OBJECTIVES

AIM AND OBJECTIVES

PRIMARY AIM

The aim of the study is to analyze the sensory nerve action potentials of two upper limb nerves (median and ulnar nerve) and two lower limb nerves (sural and common peroneal nerve) in hypothyroid patients and non hypothyroid persons. Three parameters of nerve conduction study – latency, amplitude and nerve conduction velocity are analyzed for four nerves in patients and controls.

SECONDARY AIM

To correlate the nerve conduction values with the physiological variables like age and sex of hypothyroid patients.

OBJECTIVES:

1. To compare the nerve conduction study of the hypothyroid patients with controls
2. To correlate the conduction deficits with duration and severity of the disease.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORY OF NERVE CONDUCTION STUDIES: [21]

During 1850, Helmholtz recorded the measurement of nerve conduction velocity in frog by mechanically recording the muscle twitch. In 1937, Eicher published the first report of the nerve action potential by stimulating the median and ulnar nerve.

Soon after the World War II, the clinical techniques for nerve conduction studies were developed. A paper published in 1948 by Hodes, Larrabee and German regarding the nerve conduction studies. They measured motor conduction velocity in healthy nerve and showed that to be slowed in regenerating nerve after an injury.

At about the same the time, Ed Lambert and his colleagues started to study the electrophysiological properties of diseased human muscle and nerve at the Mayo clinic. An abstract appeared in 1950 on 'Unipolar electromyograms of patients with dermatomyositis'.Arnold Carmicheal set up

a research unit after the World War II, at The National Hospital for Nervous Diseases. Carmicheal and Goerge Dawson are remembered for the advances they made in recording nerve action potential. In 1949 Dawson and Scott published their method for recording the sensory nerve action potentials.

By giving the electrical stimulation to the peripheral nerve through the skin, Eichler recorded the sensory nerve action potentials. Just because of Dawson and Scott there was a remarkable clinical development of this method.

Roger Gilliatt and Tom Sears set up a routine recording laboratory in 1955, with the equipment constructed by Bert Morton. By using the photographic superimposition of traces to improve signal to noise ratio, Sensory nerve action potential was first recorded.In patients with peripheral nerve lesions sensory nerve action potentials was reported by Gilliatt and Sears in 1958.

THE HERITAGE OF THE THYROID[3]

The ancient Greeks called as ‘bronchocele’ (tracheal out pouch) for the goitrous swelling in the neck till 19th century despite the thyroid gland was discovered 200 years earlier.

The modern name arouse in 1656, as Thomas Wharton called it as thyroid gland. Graves described four patients with features of palpitation, exophthalmoses and goiter. But his description was not widely known on European continent. Besedow was considered to be the first to describe the illness when he reported a patient in 1840. So ‘Basedow’s disease’ is the term still used by many Europeans rather than Grave’s disease.

Hypothyroidism is a clinical syndrome recognized even later than hyperthyroidism. In the 1870s, in London, hypothyroidism was named myxedema because of the swollen skin (edema) and excess content of mucin in it.

NEUROLOGICAL EFFECTS OF THYROID HORMONES[22]

The thyroid hormones are important for normalfoetal and neonatal brain development. These hormones regulate the neuronal proliferation and differentiation, myelinogenesis, neuronal growth and synapse formation. The critical period of brain development begins in utero and extends to approximately 2 years during which the thyroid hormone deficiency results in structural and physiological impairment. Hormone replacement therapy beyond this period cannot reverse the damage. Congenital hypothyroidism may result in severe and irreversible brain damage. In most of the countries including India neonatal thyroid screening program was implemented. Thyroid hormones also affect the neurological function subsequent to the critical period, but these changes can be reversed by correcting the thyroid disorder. Hypothyroidism can result in psychological disturbances, leading to myxoedema coma.

Ellen Crushell and William Reardon[23] presented a case of 23 months old boy with developmental delay, hypotonic and irritability with inconsolable cry. His TSH value was normal while screened for congenital

hypothyroidism on day 4 after his birth. Hypothyroidism was diagnosed at 4 months of age as the TSH was mildly elevated at that time with free T_4 just below normal. Later he was diagnosed as the Allan-Herndon-Dudley syndrome of X-linked disorder caused by mutations in a thyroid hormone transporter gene.

EFFECT ON NERVOUS SYSTEM OF ADULT[24]

Thyroid hormones are essential for the normal functioning of the brain and nervous system. T_4 increases the wakefulness, alertness, responsiveness to various stimulus, auditory sense, and awareness of hunger, memory and learning capacity. Hippocampus in the brain is sensitive to thyroid hormones and is necessary for some form of learning and memory. So hypothyroidism will have the following features:

- Loss of intelligence
- Impairment of memory
- Somnolence
- Slowness of sleep

- Mental and physical lethargy
- Eventually psychosis (myxoedematous madness)

SYMPATHETIC NERVOUS SYSTEM[24]

Thyroid hormones have the effects like adrenergic catecholamines as follows: increase in metabolic rate, heat production, heart rate, motor activity and central nervous system excitation. These effects are by increasing the levels of cAMP. After T_4 administration, in plasma, muscle and urine levels of cAMP are increased. The catecholamine and T_4 potentiate the action of each other.

ACTIONS ON SKELETAL MUSCLE[24]

Thyroid hormones have direct action on muscle. They increase both the electrogenic Na-K pump and the resting membrane potential. They increase the rate and amount of calcium uptake in the sarcoplasmic reticulum, thereby increasing the availability of calcium on stimulation. They increase the myosin ATPase activity. The maximal shortening of the conduction velocity found to be increased after thyroid hormone

administration. Myopathies are common in both hypothyroidism and hyperthyroidism. In hypothyroidism muscle weakness, wasting and fatigability are common. This is most often seen in the proximal muscles of the limbs and can lead to difficulties in climbing up the stairs.

NERVE CONDUCTION STUDY[25]

Nerve conduction studies provide the greatest help in assessing the peripheral nerve disorder. This study most often not only confirms the clinical diagnosis, but also gives valuable information to:

1. Exclude other suspected disorder

2. Localize focal abnormalities along a nerve

3. Define severity with objective measurements

4. Identify anomalous innervations

BASICS OF ELECTRODIAGNOSTIC SIGNALS[25]

The clinical electro diagnosis involves the recording , display, measurement and interpretation of action potentials arising from central nervous system(

evoked potentials), peripheral nerves (nerve conduction studies), and muscles(electromyography). In clinical neurophysiology, the action potential amplitude is expressed in milli-volt(mV); current in milli-amperes(mA) and time measurement in milliseconds(ms).In electro diagnostic tests, three electrodes (active, reference and ground) are used. The action potentials are measured between active and reference electrodes and the ground electrode serves as a zero voltage reference point. Two types of electrodes are used named surface and needle electrodes.

PRINCIPLE OF NERVE CONDUCTION STUDY[25]

In nerves conduction study an external stimulation was given on the surface of the skin lying over the concerned nerve to be studied. That will initiate depolarization simultaneously in all the axons of the nerves to produce a recordable response in the form of action potentials. By stimulating the nerve at two different points the response is recorded. It involves the study of both motor and sensory conduction. The strength and speed of the electrical impulses conducted along the peripheral nerve is measured by the nerve conduction study.

A bipolar stimulator is placed on the skin that lies over the anatomic course of the nerve and the impulse is generated. The intensity and duration of the stimulus is increased until all the axons of the nerve get depolarized, that results in action potential that travels down the nerve to the site of recording.

SENSORY NERVE CONDUCTION[25]

Sensory nerve conduction studies are done by placing the recording electrode on the skin lying over the particular nerve at some distance away from the stimulation site. As action potential propagates between these bipolar recording electrodes SNAP wave form is recorded and which will get displayed on the screen of the EMG recording device. It can be measured either orthodromically or antidromically. For orthodromic conduction, distal part of the nerve is stimulated and sensory nerve action potential is recorded at the proximal site of the nerve under study. For antidromic nerve conduction study, the proximal point of the nerve is stimulated and recording is done on the distal part of the same nerve.

MOTOR NERVE CONDUCTION STUDY[25]

In motor nerve conduction studies, the active electrode is placed over the motor point, which is usually at the midpoint between the origin and insertion of the muscle. The reference electrode is placed on the tendon. The distance between the active and reference electrodes influences the amplitude of CMAP(Compound muscle action potential). So the distance between these two electrodes were standardized as inter electrode distance of 3 – 4 cm. A biphasic action potential with initial negativity is recorded. Surface stimulation of healthy nerve requires a square wave pulse of 0.1ms duration with an intensity of 5 – 40 mA. In a diseased nerve, the nerve excitability is reduced and the current requirement is higher than normal.

The measurements of motor nerve conduction study include the onset latency, area, duration and amplitude of CMAP (compound muscle action potential) and nerve conduction velocity.

HYPOTHYROIDISM AND NERVOUS SYSTEM

GiroudM et al[22], done a prospective study in neonates with congenital hypothyroidism from infant specialty hospital at France. They observed a large

reduction in the action potential amplitude of the sural nerve and reduced Hofmann's reflex in neonates before starting treatment. It was transient and disappeared after hormone replacement therapy for six months.

Marcia W et al[19] studied 16 patients with primary hypothyroidism. The ENMG and NCS findings are:

- Median nerve Motor Latency prolonged (patients mean SD: 3.5) when compared with control group (mean SD: 2.7).Reduced motor amplitude and motor conduction velocity were noted.
- Ulnar nerve sensory conduction velocity reduced.
- Peroneal motor latency prolonged; motor conduction velocity reduced.
- Ulnar motor amplitude reduced; no significant changes in latency and conduction velocity.
- Sural nerve sensory conduction velocity reduced; no other significant changes.

Eslamian F et al**[18]** evaluated the signs and symptoms of neuromuscular dysfunction in primary hypothyroid patients. They found that the patients with hypothyroidism had the clinical features of mononeuropathy, proximal muscle weakness and sensorimotor polyneuropathy. The electromyography and nerve

conduction study was done in the clinic of the Tabriz University of Medical Sciences[18] at Iran and reported 45% of the patients found to be with decreased or absent deep tendon reflexes, 15%(6 cases) with neuropathy which include 4 sensory and 2 sensorimotor neuropathy.7.5% patients with myopathy and 32.5% with carpal tunnel syndrome. In hypothyroidism, there is decrease in protein degradation which is responsible for decrease in the oxidation and the glycogen deposits, which will result in energy deficit. The production of energy during aerobics, in the form of ATP, is due to the stimulation of mitochondrial respiratory activity by the thyroid hormone, under physiological conditions. It increases the ATP dependent sodium potassium pump activity. In hypothyroidism the deficient ATPase activity and ATP is associated with altered pump dependent axonal transport.

Shirabbe et al[10] studied hypothyroid patients and showed the segmental demyelination and onion bulb formation in sural nerve with scanty mucinous deposits and significant loss of large myelinated nerve fibers. The patient with primary myxoedema presented with parasthesia in hands due to carpal tunnel syndrome which in turn is due to myxedematous tissue beneath the transverse

carpal ligament. Apart from the above findings delayed sensory nerve conduction and decreased deep tendon reflexes were other findings of the same patient. Interestingly the sensory polyneuropathy was markedly reversed with hormone replacement therapy.Peripheral neuropathy was considered when three or more parameters were found abnormal in at least three different nerves.

In 1961 Nickel at al[16] did the microscopic examination of the peripheral nerves of the myxoedema patients. They found focal degenerative changes of the myelin sheath.

NEUROMUSCULAR FINDINGS IN HYPOTHYROIDISM

A retrospective study[13] done during 1980 shows the prevalence of neuropathy in hypothyroid patients ranges from 10% to 70 % and myopathy between 20 % and 80%. Another study done by Ruurd F Duyff[13]and colleagues showed about 40% of the hypothyroid patients predominantly had sign of a sensorimotor axonal neuropathy early days of hypothyroidism. This study revealed that:

- Neuromuscular complaints 79%

- Clinical weakness 38%
- Sensorimotor axonal neuropathy 42%
- Carpal tunnel syndrome 29%

Somay G et al[26] found an increase in the median motor distal latency and the median distal latency ($p<0.0001$), the reduction in the median sensory action potential amplitude ($p<0.001$) and the slowed nerve conduction velocity ($p<0.01$).

MORE COMMONLY AFFECTED NERVES IN HYPOTHYROIDISM

Median nerve entrapment at the wrist is the one of the most common neurological features in hypothyroidism. It is due to the deposition of the mucinous material in the tissues surrounding the nerve. Dyck and Lambert[9] suggested that the peripheral neuropathy is because of the metabolic alterations caused by endocrine disorders. They came to this conclusion after they studied the cases morphologically and neurophysiologically**.**

A.K. Thacker[27] et al showed complete nerve conduction block along the right and left peroneal nerves in the knee-angle segments. But there was a great response of the patient to the thyroid replacement treatment.

SURAL NERVE CONDUCTION STUDY

Paola Penza et al[20] have done nerve conduction study on a hypothyroid patient and demonstrated mild sensory neuropathy and decreased sural nerve conduction velocity (NCV) and reduced sensory nerve action potential (SNAP) amplitude. The values are 7.7μV; 36.9 m/sec on right side and 7.9 μV& 37m/sec on left side by antidromic technique: normal values 10 μV and 42m/sec.

Yuksel et al[28] studied SEP (Somatosensory evoked potential) and Blink reflex in newly diagnosed and untreated thyroid disease patients. According to this study, overall central nervous system involvement is found in 78% of patients. The metabolic alterations caused by hormonal imbalance affect Schwamm cell. Initially the function of the nerve only gets affected, but later on the structural alteration develops.

Most commonly involved nerve was the sural nerve and Median nerve sensory fibers. CTS (Carpal tunnel syndrome) is caused by mucinous materials in the tissues surrounding median nerve with hypothyroidism induced demyelination. The incidence of CTS in this study was 50%.

NERVE CONDUCTION STUDIES IN CRETINS

Moosa A, and Dubowitz, V. (1971), [8] studied the ulnar and posterior tibial nerve conduction velocities in cretins and found that the 4 out of 6 untreated cretins had reduced conduction velocity values for both the nerves. In those remaining two patients one had below normal range values (26.7 to 34 m/sec) during the first one year; and the normal value is 50.9 +/-5.4m/sec for the ulnar nerve conduction velocity and the other one had reduced posterior tibial nerve conduction velocity.

SUBCLINICAL HYPOTHYROIDISM AND NEUROPATHY

Magri F et al [29] reported the intra epidermal nerve fiber density reduction as a marker of preclinical asymptomatic small-fiber sensory neuropathy. They took

the skin biopsy from the upper thigh and distal leg and did the nerve conduction study. Measurement of nerve fiber density was done by immunoflurescence technique and found out that 60% of the patients with OH (Overt Hypothyroidism) had reduced IENF (Intra Epidermal Nerve Fiber) density at distal leg and 20% at the proximal site. An abnormal IENF density was found at the distal end in 25% cases and proximal leg in 12.5% of patients. They came for a conclusion that a considerable number of untreated hypothyroid patients may have pre clinical asymptomatic small-fiber sensory neuropathy.

SCREENIG FOR THYROID DISEASE IN PATIENTS WITH CARPAL TUNNEL SYNDROME – VALUABLE OR NOT.

Suresh E and Morris IM[30] performed screening for hypothyroidism and found two patients had carpal tunnel syndrome. The incidence was 1.5% in this study.

Lai CL et al[31] studied the central and peripheral nerve conduction in patients with primary hypothyroidism and the effect of thyroxine treatment on the nerve conduction velocities. Before treatment, SSEP (Somato-Sensory Evoked Potential) BAEP (Brain Stem Auditory evoked potential) and VEP (Visual Evoked Potential) latencies were significantly delayed in 11/20 patients. Regarding the peripheral nerve conduction, decreased conduction velocities and diminished amplitudes were found in fourteen patients. They proved that there was a marked improvement in both central and peripheral nerve conduction after treating the patients with thyroxine for 6 months duration.

Cruz MW et al[32] reported carpal tunnel syndrome in 43.7% of the hypothyroid patients ,ENMG (Electroneuromyography) abnormality in 87.5% and myopathy in 46.6%.The most common symptom was (75%) cramps. The results of this study as follows:

- Parasthesia on hands (68.7%).
- Weakness (62.5%).
- Stiffness (43.7%).

- Ptosis (25%).
- Diplopia and muscular hypertrophy (12.5%).

Regarding nerve conduction, sensory amplitude was reduced or abolished in 68.7% of the patients. CTS was found in 43.7% of the patients. Concomitance of CTS and myopathy were seen in 25% of the patients. During the physical examination patients presented with weakness were 28.5% and muscle enzyme levels increased in 42.8%.

EttoreBeghi et[33] al compared all electrical parameters like CV (conduction velocity), distal motor latency and potential amplitude of the hypothyroid patients with the standard values. The normal limits of CV and distal latency were set at 2.5 SD from the mean values of the age-matched controls. They made definite electrophysiological diagnosis of polyneuropathy in 28 cases (72%). Other findings of this study are:

- Distal latency was most commonly impaired in the peroneal nerve (36%)
- Motor and sensory action potential amplitude was decreased in 26% of the patients.

RaffaelloNemni et al[34] reported about the clinical, electrophysiological and morphological findings in four hypothyroid cases. Ulnar and deep peroneal motor conduction velocities and median and sural nerve sensory conduction velocities were measured. They used surface electrodes for the stimulation of nerve in warm room. Cutaneous temperature was maintained at 36˚C by a heating lamp. Electroneurographic examination demonstrated increased distal latencies of nerve action potentials and moderate slowing of the nerve conduction velocities. Patients with sensorimotor polyneuropathy, found to be with a distal to proximal progression that first involves the lower limbs than the upper limb. The severity of the clinical picture of the neuropathy was more often related to the duration of the disease than to the thyroid hormone deficiency. In this study all the patients had reduced motor action potential amplitude (MAP) and sensory nerve action potential (SAP) amplitude. Mild slowing of the nerve conduction velocity of both the motor and sensory nerve is due to the presence of the axonal neuropathy.

YeasminS et al[35] published an article electrophysiological and clinical finding of sensory neuropathy in hypothyroidism. They included 40 subjects of 20 to 50 years old hypothyroid patients (group B) of both the sex. 30 apparently healthy controls were enrolled in the study as controls (group A). It was carried

out in the department of physiology, Dhakaduring the period ofJanuary 2005 to December 2005. On the basis of TSH level they further divided the study group into:

- Group A :-Euthyroid control group
- Group B_1 :- Hypothyroid ;15 patients (TSH<60 mIU/L) and
- Group B_2:- Hypothyroid; 25 patients (TSH > 60 mIU/L)

The statistical analysis was by one way ANOVA. The typical clinical features of neuropathy were absent in all hypothyroid patients except hypo or areflexia of most of the deep tendon reflexes.

Nerve conduction study in the median nerve revealed significantly increased distal latency (SDL) and reduced conduction velocity (SNCV) in both of Group B1 and B2 when compared to Group A (controls).

But ulnar nerve was taken for consideration; these values differ significantly between euthyroids (Controls) and severe hypothyroid patients (Group B2). It did not show any statistical significant with group B1(less severe hypothyroid patients). The (SDL, SNCV) values of sural nerve were statistically significant

between groups A (Euthyroid) with all hypothyroids (both the groups B1 and B2). There were no significant neurophysiologic changes in two hypothyroid groups (B1& B2).

By this study, abnormal nerve conduction study of 67.5% seen in hypothyroid patients. Among them, 66% of the patients were in severe hypothyroid group (B1) and 34% of the subjects were in less severe group (B2). TheSNAP and CMAP were not included in this study, as the changes in the amplitude were very minimal.

Jane Martin et al[36] reported an interesting case of polyneuropathy with spurious polycythaemia (Gaisbock's syndrome) in hypothyroidism. This patient presented with absence of knee and angle reflexes and diminished pin prick sensation in a glove and stocking sensation. Abnormal nerve conduction study with sensorimotor neuropathy of axonal type was the finding of his electrophysiological study.

Adriana Patrica et al[37] reported the hypothyroid associated polyneuropathy in 6 dogs. They presented with exercise intolerance, generalized weakness and ataxia. All the dogs showed slow tibial motor nerve conduction velocities

MATERIALS

AND

METHODOLOGY

MATERIALS AND METHODS

After obtaining clearance from Institutional Human Ethical Committee (IHEC) at PSG IMS &R, the study was started in patients attending Medicine, Endocrinology and Neurology outpatient department of our hospital.

It included 30 cases of hypothyroid patients, of both sexes between the age group of 20 to 60 years. The controls were selected from patients who do not have the thyroid hormone deficiency and attending the medicine and neurology OPD of both sexes of the same age group as cases 20 – 60 years.

INCLUSION CRITERIA:

1. Age 20-60 yrs
2. Hypothyroid patient(TSH >10 mIU/L)
3. Both male and female

EXCLUSION CRITERIA:

1. Other possible causes of neuropathy or neuromuscular disease like, DM, Alcoholism, Liver and kidney disease.
2. Family history of neuropathy
3. Users of drugs that causes neuropathy.
4. Pre-existing neuropathy.

The purpose of this study and the procedure was explained in their mother tongue to all the subjects included. The informed consent was obtained from them. Detailed history regarding relevant complaint, duration of the disease and the treatment were obtained from each. A general physical examination in detail was done. Patients were checked for hypertension and peripheral nerve thickening and relevant clinical findings obtained. A detailed systemic examination was done during routine neurological examination. Recent thyroid function test results were collected for all hypothyroid patients included in our study. The nerve conduction study was done to volunteers.

The protocol is attached in annexure that contains patient data, history, examination and investigations.

EQUIPMENT

The nerve conduction studyis done using Recorders Medicare System (RMS) EMG EPM2K version-1(PHOTO 1). There are three types of electrodes, in nerve conduction studies,the surface electrodes made up of silver chloride and nickel are used frequently. The electrode jelly is applied over the skin after cleaning the skin and surface electrodes are applied over the jelly, which gives an interface between the skin and the equipment.

PROCEDURE OF NERVE CONDUCTION STUDY[17]

1. The patient is made to lie down on the couch. Clean the skin before applying the jelly and electrodes
2. Fix the surface electrode over the skin which is on the nerve and supplying muscle
3. Connect the electrode through the pre amplifier to the oscilloscope

4. Keep the sweep at 5 ms/cm.
5. By the stimulating electrodes, the nerve is stimulated at the distal end and we monitor the action potential on the oscilloscope. The interval between the beginning of the stimulus artefact and the first deflection of the action potential is measured, gives the latent period.
6. The action potential is recorded by stimulating the proximal end of the nerve. The difference between the two latent periods will give the time taken by the impulse to travel from the proximal to the distal end.
7. Measure the distance between the points of stimulation

The median, ulnar , peroneal and sural nerves are selected for our study.

MEDIAN SENSORY NERVE CONDUCTION[17, 38]

It is measured by arthodromic stimulation of the nerve. Three centimeter proximal to the distal wrist, the recording electrode is placed. The reference electrode is placed at a 3cm distance proximally. The ring electrodes are placed at the second or third digits for stimulation. The cathode is placed at first interphalangeal joint and anode 3cm distal. The distal latency, sensory

nerve action potential and conduction velocity of different segments are calculated.

ULNAR NERVE CONDUCTION VELOCITY[17, 38]

By arthodromic stimulation the sensory nerve conduction velocity of the ulnar nerve was measured. The ring electrodes connected to the interphalangeal joint of the fifth digit is stimulated. The sensory nerve action potential can be record at various sites along the course of ulnar nerve.

SURAL NERVE CONDUCTION STUDY[17, 38]

Sural nerve is derived from S1 and S2 roots and formed by two components. The medial part is derived from tibial nerve and lateral component from peroneal nerve. Sural is purely sensory nerve. The surface electrode between lateral malleoulus and tendoachilles records the sural nerve conduction velocity. By antidromical stimulation of the nerve at 10 -16 cm proximal to the recording electrode , distal to the lower border of the gastronemius at the junction of the middle and lower third of the leg. The leg should be relaxed fully during recording, so the lateral position is convenient.

SUPERFICIAL PERONEAL NERVE CONDUCTION VELOCITY[17, 38]

It can be recorded by placing the active electrode just above the junction of lateral third of a line connecting the malleoli and reference electrode 3 cm distal to it.

ONSET LATENCY[17, 38]

The onset latency is the time in milliseconds from the stimulus artifact to the first negative deflection of CMAP. For better visualization of the take off, the latency should be measured at a higher gain than the one used for CMAP amplitude measurement.

The onset latency is a measure of conduction in the fastest conducting motor fibers. It also includes the neuromuscular transmission time and the propagation time along the muscle membrane which constitute the residual latency.

THE AMPLITUDE[17, 38]

The amplitude of the CMAP is measured from the base line to the negative peak (base to peak) or between negative and positive peaks (peak to peak). The amplitudes correlate with the number of nerves fibers.

THE DURATION[17, 38]

The duration of CMAP is measured from the onset to the negative or positive peak or the final return of wave form to the base line. The duration correlates with the density of small fibers. The area under the CMAP also measured and computer analysis done.

NERVE CONDUCTION VELOCITY[17, 38]

Motor nerve conduction velocity is calculated by measuring the distance in millimeter between two points of stimulation, which is divided by the latency difference in milliseconds. The nerve conduction velocity is expressed as m/s. Measurement of latency difference between the two points of stimulation eliminates the effect of residual latency.

Nerve Conduction Velocity = Distance (mm) / Proximal – Distal Latency (m/s)

D is the distance between proximal and distal stimulation in millimeter.

PRECUATIONS[17, 38]

1. Proper instructions should be given to the subject and motivate to provide cooperation
2. Ask the subject to be relaxed
3. The recording place should be quiet, comfortable and provide privacy
4. Ground the subject properly.

VARIABLES AFFECTNG THE STUDY[17, 38]

It may be physiological variables like age, sex, height, temperature ortechnique.

AGE

Conduction velocities change significantly with advancing age of the subject. Slow conduction velocities were recorded over the age of 60 years. That is why we excluded the patients above 60 years from our study.

HEIGHT

Conduction velocities in the legs slows with height.

SEX

Gender differences are primarily caused by differences in height.

TEMPERATURE

Low temperature results in slow conduction velocities but nerve and muscle action potential amplitudes are higher.

LOWERLIMB VERSUS UPPER LIMB

The upper limb nerve conduction velocity (for median and ulnar nerve) is faster if compared with the tibial and peroneal.

TECHNICAL BIAS[17, 38]

The technical errors are common in motor and sensory nerve conduction studies. Any unexpected finding should be considered to be caused by a technical error until proven otherwise.

1. Spread of current because of excess stimulation.

2. Small responses due to sub maximal stimulation.

3. Distance measurement errors.

4. Incorrect limb positioning.

5. Incorrect location of the recording electrodes.

6. Failure of the stimulating system.

7. Faulty connection in the recording system.

8. Spread of stimulating current to an adjacent nerve or root not under the study.

9. Anomalous crossover between the nerves: anomalous innervations of the muscle can also result in errors of amplitude measurement.

RISKS[17, 38]

- Nerve conduction studies are essentially risk free in normal subjects.
- It is contraindicated in subjects with intra cardiac catheters and also in patients with pacemakers. We did not include any such critical patients in our study.

NORMAL VALUES FOR MOTOR NCS[17]

NERVE	LATENCY(ms)	AMPLITUDE(mA)	NERVE CONDUCTION VELOCITY(m/s)
Median nerve			
Wrist	3.77 ±0.4	8.1±2.62	
Elbow	7.62±0.65	7.84±2.25	58.52±3.76
Ulnar nerve			
Wrist	2.59±0.39	5.7±2	
Elbow	6.1±1.69	5.5±2	58.7±5.1
Common peroneal nerve	4.70±1.5	8.8±2.5	49.0±3.4

NORMAL VALUES FOR SENSORY NCS[17]

NERVE	LATENCY(ms)	AMPLITUDE(mA)	NERVE CONDUCTION VELOCITY(m/s)
Median nerve	3.06± 0.41	38.5±15.6	56.20±5.80
Ulnar nerve	2.83±0.4	35.0±14.7	54.17±9.4
Sural nerve	2.56±0.61	18.0±10.5	50.9±5.4

PHOTO 1

Recorders Medicare System (RMS) EMG EPM2K version-1

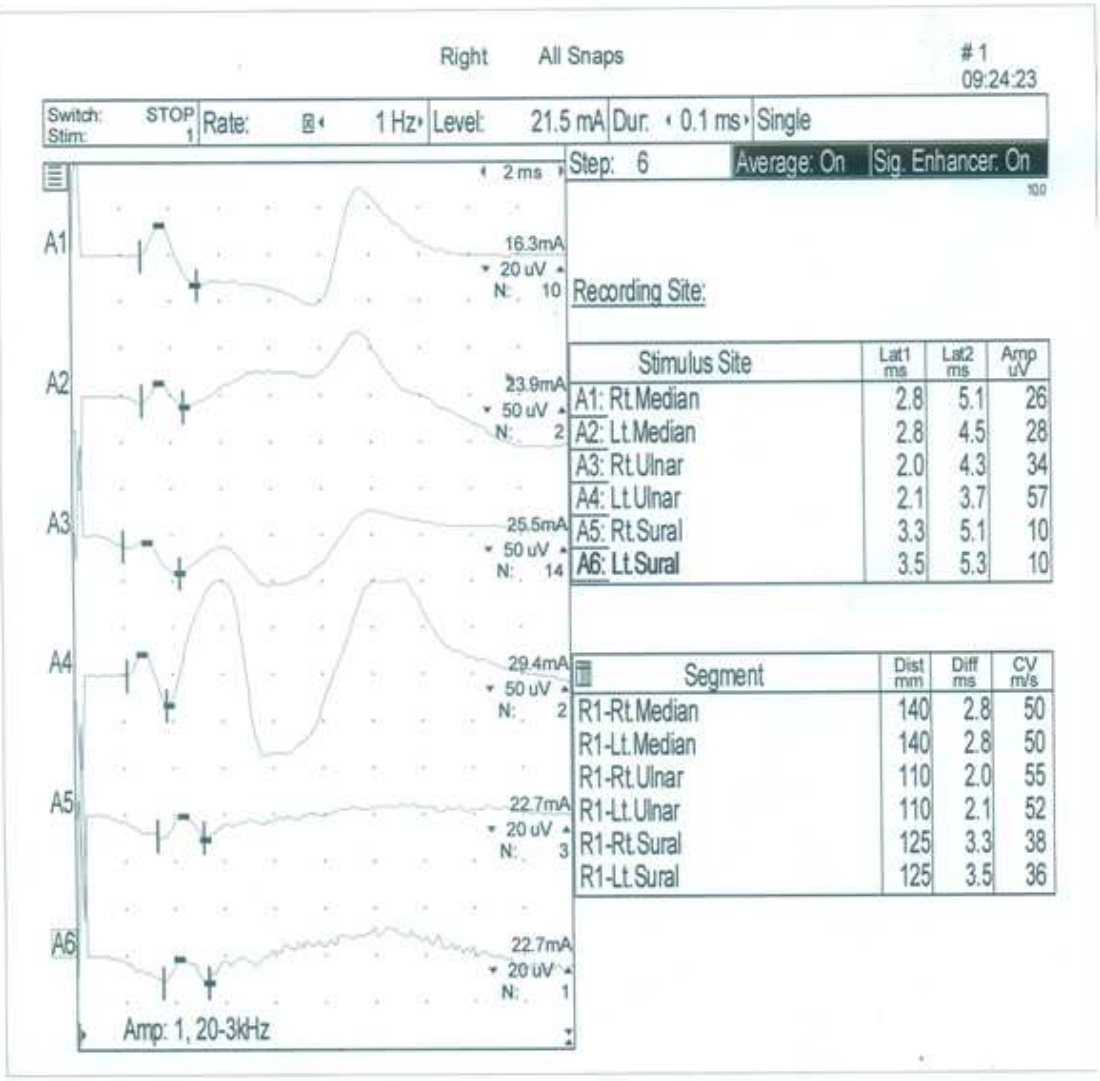
PHOTO 2

NERVE CONDUCTION STUDY

NERVE CONDUCTION RESULT

File Id: v0024 mbp Session: 2014-03-18 19 Mar 14 09:24

Nicolet VikingQuest VIASYS Healthcare NeuroCare



Recording Site:

Stimulus Site	Lat1 ms	Lat2 ms	Amp uV
A1: Rt.Median	2.8	5.1	26
A2: Lt.Median	2.8	4.5	28
A3: Rt.Ulnar	2.0	4.3	34
A4: Lt.Ulnar	2.1	3.7	57
A5: Rt.Sural	3.3	5.1	10
A6: Lt.Sural	3.5	5.3	10

Segment	Dist mm	Diff ms	CV m/s
R1-Rt.Median	140	2.8	50
R1-Lt.Median	140	2.8	50
R1-Rt.Ulnar	110	2.0	55
R1-Lt.Ulnar	110	2.1	52
R1-Rt.Sural	125	3.3	38
R1-Lt.Sural	125	3.5	36

RESULTS

RESULTS

Three parameters (latency, amplitude and nerve conduction velocity) of motor component of three nerves (Median nerve, Ulnar nerve and Peroneal nerve) and sensory component of three nerves (Median nerve, Ulnar nerve and Sural nerve) were compared between cases (Hypothyroidism) and controls (non hypothyroid). The physiological data (age, gender and duration of disease) were correlated with nerve conduction values, considering sum of amplitudes and sum of Nerve conduction velocities. Statistical analysis was done using SPSS software by unpaired 't' test and ANOVA for various analysis.

COMPARISON OF MOTOR COMPONENT OF EACH NERVE:

COMPARISON OF MOTOR NERVE CONDUCTION VALUES BETWEEN HYPOTHYROID PATIENTS AND NON HYPOTHYROID CONTROLS

Analysis was done by unpaired students't' test. The p value <0.05 was considered to be statistically significant

COMPARISON OF MEDIAN NERVE OF HYPOTHYROID PATIENTS WITH EUTHYROID CONTROLS

RIGHT MEDIAN NERVE (Table 1 and chart 1)

Theproximal latency of median nerve in controls was 3.45±0.38and in cases was 3.63± 1.03. The increase in the latency in cases was not statistically significant with p value of 0.39.

The distal latency of median nerve inthe controls was 7.80±0.58 and that of the cases was 7.44±1.28. The decrease in distal latency in cases was not statistically significant with p value of 0.16.

Themotor action potential amplitude of median nerve in the controls was 9.92±1.97 and that of the cases was 10.42±3.14.The increase in the amplitude in cases was not statistically significance with p value of 0.46.

The nerve conduction velocity (NCV) of median nerve in controls was 58.50±4.59 and that of cases was 46.09±8.61. The decrease in NCV in cases was highly significant with p value < 0.001.

LEFT MEDIAN NERVE(Table 2 and chart 2)

The proximal latency of median nerve in the controls was 3.31±0.33 and that of cases was 3.51±1.13.The increase in the proximal latency in cases was not statistically significant with p value of 0.37.

The distal latency of median nerve inthe controls was 7.62±0.46 and that of the cases was 7.32±1.41. The decrease of the distal latency in the cases was not statistically significance with p value of 0.27.

The compound motor action potential amplitude of median nerve in the controls was 11.66±3.32 and that of the cases was 12.09±3.94. The increase in the action potential in cases was not statistically significant with p value of 0.64.

The nerve conduction velocity (NCV) of median nerve in the controls was 58.66±5.02 and that of the cases was 60.52±4.77. The increase in the nerve conduction velocity of the median nerve in the cases was not statistically significant with p value of 0.14.

COMPARISON OF ULNAR NERVE OF HYPOTHYROID AND EUTHYROID SUBJECTS

RIGHT ULNAR NERVE (Table 3 and chart 3)

The proximal latency of ulnar nerve in the controls was 2.17 ±0.38 and that of cases was 2.28 ±0.27. The increase in the latency in cases was not statistically significant with p value of 0.35.

The distal latency of ulnar nerve in the controls was 6.56 ±0.79 and that of the cases was6.69 ±0.58. The increase in the latency in cases was not significant with p value of 0.37

The amplitude of ulnar nerve in the controls was 11.77±2.43 and that of the cases was 12.66±3.55. The increase in the amplitude in the cases was not statistically significant with p value of 0.26.

The nerve conduction velocity (NCV) of ulnar nerve in controls was 59.65±7.15 and that of the cases was 53.65±4.02. The decrease in NCV in cases was highly significant with p value <0.001.

LEFT ULNAR NERVE (Table 4 and chart 4)

The proximal latency of ulnar nerve in the controls was 2.32±0.43 and that of the cases was 2.22±0.29. The decrease in the proximal latency in the cases was not statistically significant with p value of 0.30.

The distal latency of ulnar nerve in the controls was 7.03±0.63 and that of the cases was 6.51±0.46. The decrease in the distal latency of the ulnar nerve in the cases was not statistically significant with p value of 0.30

The amplitude of the ulnar nerve in the controls was 12.03±2.37 and that of the cases was 11.51±2.92. The decrease in the amplitude in cases was not statistically significant with p value of 0.45.

The nerve conduction velocity (NCV) of ulnar nerve in controls was 60.12±6.96 and that the cases was 55.15±5.24. The decrease in NCV in cases was significant with p value < 0.01

COMPARISON OF PERONEAL NERVE OF HYPOTHYROID AND EUTHYROID SUBJECTS

RIGHT PERONEAL NERVE (Table 5 and chart 5)

The proximal latency of peroneal nerve in the controls was 3.44±0.50 and that of the cases was3.11±0.80. The decrease in cases was not statistically significant with p value of 0.06.

The distal latency of peroneal nerve in the controls was 10.35±0.88 and that of cases was 9.77±0.80.The decrease in the latency in cases was statistically significant with p value of <0.05.

The amplitude of peroneal nerve in the controls was 6.6±1.92 and that of the cases was 6.08±2.32. The decrease in the amplitude in cases was not significant with p value of 0.34.

The nerve conduction velocity (NCV) of peroneal nerve in the controls was 47.95±95 and that of the cases was 48.59±5.59. The increase in the conduction velicity of the right peroneal nerve in the cases was not statistically significant with p value of 0.26.

LEFT PERONEAL NERVE (Table 6 and chart 6)

The proximal latency of peroneal nerve in the controls was 3.40±0.53 and that of the cases was3.41±0.42. The increase in the proximal latency of the left peroneal nerve in the case was not statistically significant with p value of 0.92.

The distal latency of peroneal nerve in the controls was 10.28 ±0.81and that of the cases was 10.72±0.96. The increase in the cases was not statistically significant with p value 0.61.

The nerve conduction velocity (NCV) of peroneal nerve in the controls was 48.90±4.74 and that of the cases was 52.96 ±7.20. The increase in the conduction velocity in case was statistical significant with pvalue < 0.05.

COMPARISON OF SENSORY NERVE CONDUCTION VALUES BETWEEN HYPOTHYROID PATIETNS AND NON HYPOTHYROID CONTROLS

Analysis done by unpaired students‘t’ test. The p value <0.05 was considered to be statistically significant.

COMPARISON OF (SENSORY) MEDIAN NERVE OF HYPOTHYROID PATIENTS WITH EUTHYROID CONTROLS

RIGHT MEDIAN NERVE (Table 7 and chart 7)

The latency of median nerve in the controls was 2.38±0.35 and that of the cases was 2.71±1.06. Theincrease in the case was not statistically significant with p value of 0.10.

The amplitude of median nerve in the controls was 61.74±30.63 and that of the cases was 46.80±25.83. The decrease in the amplitude in cases was significant with p <0.05.

The nerve conduction velocity (NCV) of median nerve in the controls was 61.03±10.89 and that of the cases was 55.23±14.88. The decrease in the cases was not statistically significant with p value of 0.90.

LEFT MEDIAN NERVE (Table 8 and chart 8)

The latency of median nerve in the controls was 2.29 ±0.47 and that of the cases was2.65±1.32. The increase in the cases was not statistically significant with p value of 0.17.

The amplitude of median nerve in the controls was 58.23±29.25 and that of the cases was 62.23±25.13. The increase in the cases was not statistically significant with p value of 0.052.

The nerve conduction velocity (NCV) of median nerve in the controls was 62.76±10.94 and that of the cases was 58.26±16.17. The decrease in the conduction velocity in the cases was not statistically significant with p value of 0.21.

COMPARISON OF ULNAR NERVE OF HYPOTHYROID AND EUTHYROID SUBJECTS

RIGHT ULNAR NERVE (Table 9 and chart 9)

The latency of ulnar nerve in the controls was 1.87±0.26 and that of the cases was 1.71±0.29. The decrease in the latency in cases was not statistically significant with p value 0.08.

The amplitude of ulnar nerve in the controls was53.53±28.12 and that of the cases was 65.53±36.33. The increase in the amplitude in cases was not statistical significant with p value of 0.15.

The nerve conduction velocity (NCV) of ulnar nerve in the controls was64.10±29.49 and that of the cases was 63.36±9.49. The decrease in the conduction velocity in the cases was not statistically significant with p value of 0.15.

COMPARISON OF ULNAR NERVE OF HYPOTHYROID AND EUTHYROID SUBJECTS

LEFT ULNAR NERVE (Table 10 and chart 10)

The latency of ulnar nerve in the controls was 1.91 ±0.42 and that of the cases was 1.71±0.54. The decreased latency of ulnar nerve in the cases was not statistically significant with p value of 0.11.

The amplitude of ulnar nerve in the controls was 48.53±2.12 and that of the cases was 55.00±3.33. The increase in the amplitude of the ulnar nerve in the cases was not statistically significant with p value of 0.08.

The nerve conduction velocity (NCV) of ulnar nerve in the controls was 57.63±10.15 and that of the cases was65.76±14.5. The increase conduction velocity in case was not statistically significant with p value of 0.05.

COMPARISON OF SURAL NERVE OF HYPOTHYROID AND EUTHYROID SUBJECTS

RIGHT SURAL NERVE (Table 11 and chart 11)

The latency of sural nerve in the controls was 2.44±0.67 and that of the cases was 2.53±0.73. The increase in the latency in cases was not statistically significant with p value of 0.60.

The amplitude of sural nerve in the controls was 12.28±7.48 and that of the cases was 13.30±10.79. The increase in the amplitude in cases was not statistically significant with p value of 0.67.

The nerve conduction velocity (NCV) of sural nerve in the controls was 56.70±22.11 and that of the cases was 50.64±14.93. The decrease in cases was not statistically significant with p value of 0.22.

LEFT SURAL NERVE (Table 12 and chart 12)

The latency of sural nerve in the controls was 2.44±0.57 and that of the cases was 2.44 ±0.52. There was no statistical significance as the p value was 0.99.

The amplitude of sural nerve inthe controls was 18.28±18.52 and that of the cases was 13.34±8.19. The decrease in cases was not statistically significant with p value of 0.18.

The nerve conduction velocity (NCV) of sural nerve incontrols was 56.10±13.47 and that of the cases was 45.23±3.97. The decrease in the sural nerve conduction velocity in cases was statistically significant with p value < 0.001.

COMPARISON OF AGE OF THE HYPOTHYROID PATIENTS WITH SUM OF NERVE CONDUCTION VELOCITES.

Cases were divided into two age groups 20 –40 and 41 - 60 groups. Analysis done by unpaired students‘t’ test.

Age group 20-40: 18 patients and

Age group 41-60years: 12 patients

The nerve conduction velocity in the patients under age group 20-40 years was 56.71±6.25 and that of the patients under the age group 41-60 years was 62.56±15.71. There was no statistically significant (p value of 0.41) difference between two groups, considering sum of amplitudes.

COMPARISON OF DURATION OF DISEASE IN HYPOTHYROID PATIENTS WITH SUM OF NERVE CONDUCTION VELOCITES.

Cases were divided into three age groups as follows:

Group 1: Newly diagnosed patients. There were 5 patients in group 1.

Group 2: duration of disease up to 5 years (19 patients)

Group 3: duration of disease >5years to 10years (6 patients).

The nerve conduction velocity in the patients under age group 1 was54.63±4.56. The nerve conduction velocity of the patients under

the group 2 was56.98±6.58 and that of group 3 was57.23±6.75.There was no statistically significant difference between the group 1 and group2 with p value 0.36 , considering sum of conduction velocities. The group 1 and group 3 were compared and it did not show any significant with p value 0.89. The group 2 and group 3were compared, there was no statistical significance with p value 0.17.

THE PERCENTAGE OF MEDIAN, ULNAR, SURAL AND COMMON PERONEAL NERVE INVOLVEMENT IN HYPOTHYROIDISM

In 30 hypothyroid patients and 30 controls all the 3 parameteres were considered for each nerve (median, ulnar, sural and CPN), and if one parameter was abnormal, the nerve was considered to be involved.

RESULTS OF NERVE CONDUCTION STUDIES IN PATIENTS:

MEDIAN NERVE-MOTOR

11 patients had abnormal nerve conduction values.

36.6% of median nerve was affected in hypothyroidism.

MEDIAN NERVE-SENSORY COMPONENT

5 patients had abnormal nerve conduction values.

16.6 % of median nerve was affected in hypothyroidism.

ULNAR NERVE -MOTOR COMPONENT

3 patients had abnormal nerve conduction values.

10 % of ulnar nerve was affected in hypothyroidism.

SURAL NERVE

5 patients had abnormal nerve conduction values.

16.6 % of sural nerve was affected in hypothyroidism

PERONEAL NERVE

2 patients had abnormal nerve conduction values.

6% of median nerve was affected in hypothyroidism.

This study showed that median was the most affected nerve in upper limb and sural nerve was most affected in the lower limb.

THE MOST AFFECTED PARAMETERS

In those with hypothyroidism, (150 nerves studied ie, 5 nerves of 30 patients) 27 patients had prolonged latencies (18%), 5 patients had reduced amplitudes (3%) and 53 had decreased NCV (35.33%).

In controls 4 had abnormal latencies, 1 had abnormal amplitude and 5 had abnormal nerve conduction velocities. This analysis showed that the nerve conduction velocity is the most affected parameter in hypothyroid induced neuropathy.

TABLES AND CHARTS

TABLE: 1

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF RIGHT MEDIAN NERVE-MOTOR COMPONENT BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P Value
Proximal latency (ms)	Controls	30	3.45±0.38	0.39(NS)
	Cases	30	3.63± 1.03	
Distal latency(ms)	Controls	30	7.80±0.58	0.16(NS)
	Cases	30	7.44±1.28	
Amplitude (mV)	Controls	30	9.92±1.97	0.46(NS)
	Cases	30	10.42±3.14	
NCV (m/s)	Controls	30	58.50±4.59	< 0.001*
	Cases	30	46.09±8.61	

*** Statistically Significant**

NS-Non Significant

TABLE: 2

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF MOTOR COMPONENT OF LEFT MEDIAN NERVE BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P Value
Proximal latency (ms)	Controls	30	3.31±0.33	0.37(NS)
	Cases	30	3.51±1.13	
Distal latency(ms)	Controls	30	7.62±0.46	0.27(NS)
	Cases	30	7.32±1.41	
Amplitude (mV)	Controls	30	11.66±3.32	0.64(NS)
	Cases	30	12.09±3.94	
NCV (m/s)	Controls	30	58.66±5.02	0.14(NS)
	Cases	30	60.52±4.77	

*** Statistically Significant**

NS-Non Significant

TABLE: 3

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF MOTOR COMPONENT OF RIGHT ULNAR NERVE BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P Value
Proximal latency (ms)	Controls	30	2.17±0.38	<0.05*
	Cases	30	2.37±0.27	
Distal latency(ms)	Controls	30	6.56 ±0.58	<0.001*
	Cases	30	7.60 ±0.79	
Amplitude (mV)	Controls	30	11.77±2.43	0.26(NS)
	Cases	30	12.66±3.55	
NCV (m/s)	Controls	30	59.65±7.15	<0.001*
	Cases	30	53.65±4.02	

*** Statistically Significant**

NS-Non Significant

TABLE: 4

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF MOTOR COMPONENT OF LEFT ULNAR NERVE BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P Value
Proximal latency (ms)	Controls	30	2.32±0.43	0.30(NS)
	Cases	30	2.22±0.29	
Distal latency(ms)	Controls	30	7.03±0.63	<0.01*
	Case	30	6.51±0.46	
Amplitude (mV)	Controls	30	12.03±2.37	0.45(NS)
	Cases	30	11.51±2.92	
NCV (m/s)	Controls	30	60.12±6.96	< 0.01*
	Cases	30	55.15±5.24	

*** Statistically Significant**

NS-Non Significant

TABLE: 5

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF RIGHT COMMON PERONEAL MOTOR NERVE BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P Value
Proximal latency (ms)	Controls	30	3.44±0.50	0.06.(NS)
	Cases	30	3.11±0.80	
Distal latency(ms)	Controls	30	10.35±0.88.	0.06(NS)
	Case	30	10.24 ±0.80	
Amplitude (mV)	Controls	30	6.6±1.92.	0.34(NS)
	Cases	30	6.08±2.32	
NCV (m/s)	Controls	30	47.95±95	0.26(NS)
	Cases	30	48.59±5.59	

*** Statistically Significant**

NS-Non Significant

TABLE: 6

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF LEFT COMMON PERONEAL MOTOR NERVE BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P Value
Proximal latency (ms)	Controls	30	3.40±0.53	0.92(NS)
	Cases	30	3.41±0.42	
Distal latency(ms)	Controls	30	10.28 ±0.81	0.61(NS)
	Cases	30	10.72±0.96	
Amplitude (mV)	Controls	30	5.68 ±1.90	0.60(NS)
	Cases	30	5.90±1.96	
NCV (m/s)	Controls	30	48.90±4.74	< 0.05*
	Cases	30	52.96 ±7.20	

*** Statistically Significant**

NS-Non Significant

TABLE: 7

COMPARISON OF LATENCY, AMPLITUDE AND NCV SENSORY COMPONENT OF RIGHT MEDIAN NERVE BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P Value
Proximal latency (ms)	Controls	30	2.38±0.35	0.10(NS)
	Cases	30	2.71±1.06	
Amplitude (mV)	Controls	30	61.74±30.63	<0.05*
	Cases	30	46.80±25.83	
NCV (m/s)	Controls	30	61.03±10.89	0.90(NS)
	Cases	30	55.23±14.88	

*** Statistically Significant**

NS-Non Significant

TABLE: 8

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF SENSORY COMPONENT OF LEFT MEDIAN NERVE BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P value
Proximal latency (ms)	Controls	30	2.29 ±0.47	0.17(NS)
	Cases	30	2.65±1.32	
Amplitude (mV)	Controls	30	58.23±29.25	0.052(NS)
	Cases	30	62.23±2513	
NCV (m/s)	Controls	30	62.76±10.94	0.21(NS)
	Cases	30	58.26±16.17	

*** Statistically Significant**

NS-Non Significant

TABLE:9

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF RIGHT ULNAR NERVE-SENSORY COMPONENT BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P value
Proximal latency (ms)	Controls	30	1.87±0.26	0.08(NS)
	Cases	30	1.71±0.29	
Amplitude (mV)	Controls	30	53.53±28.12	0.15(NS)
	Cases	30	65.53±36.33	
NCV (m/s)	Controls	30	64.10±29.49	0.15(NS)
	Cases	30	63.36±9.49	

*** Statistically Significant**

NS-Non Significant

TABLE: 10

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF LEFT ULNAR NERVE-SENSORY COMPONENT BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P value
Proximal latency (ms)	Controls	30	1.91 ±0.42	0.11(NS)
	Cases	30	1.71±0.54	
Amplitude (mV)	Controls	30	48.53±2.12	0.08(NS)
	Cases	30	55.00±3.33	
NCV (m/s)	Controls	30	57.63±10.15	0.05(NS)
	Cases	30	65.76±14.54	

*** Statistically Significant**

NS-Non Significant

TABLE: 11

COMPARISON OF LATENCY, AMPLITUDE AND NCV OF RIGHT SURAL NERVE (SENSORY) BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P value
Proximal latency (ms)	Controls	30	2.44±0.67	0.60(NS)
	Cases	30	2.53±0.73	
Amplitude (mV)	Controls	30	12.28±7.48.	0.67(NS)
	Cases	30	13.30±10.79	
NCV (m/s)	Controls	30	56.70±22.11	0.22(NS)
	Cases	30	50.64±14.93	

*** Statistically Significant**

NS-Non Significant

TABLE: 12

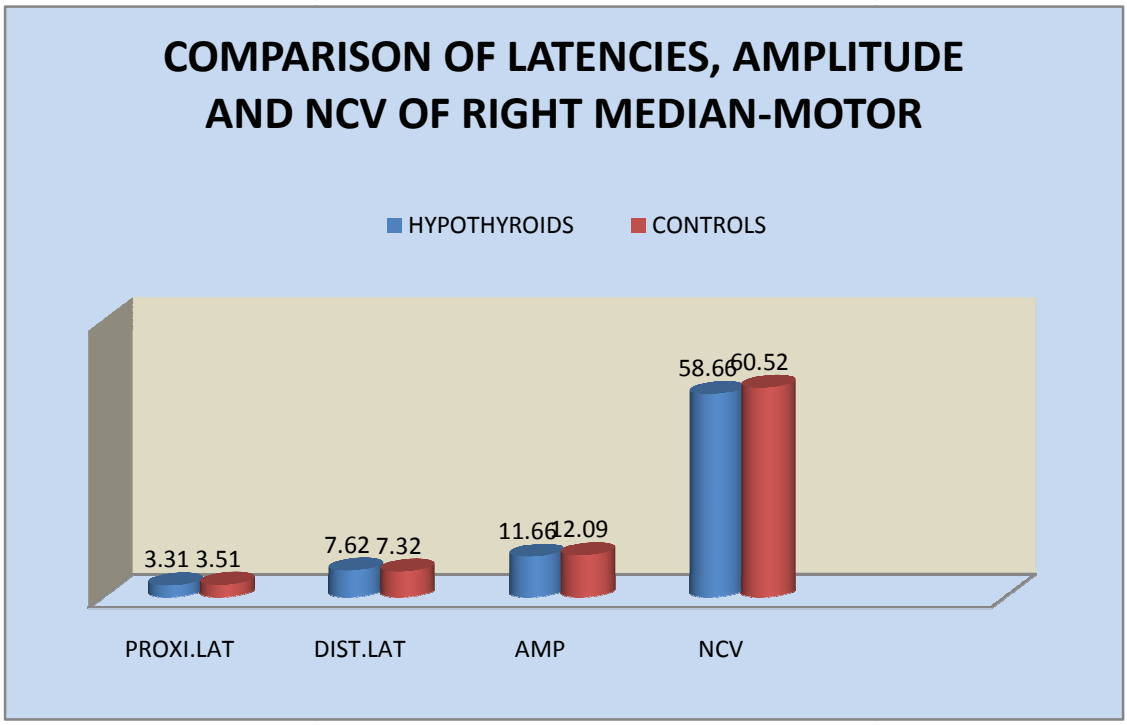
COMPARISON OF LATENCY, AMPLITUDE AND NCV OF LEFT SURAL NERVE (SENSORY) COMPONENT BETWEEN HYPOTHYROID PATIENTS AND CONTROLS

PARAMETER	GROUP	NUMBER	MEAN±SD	P value
Proximal latency (ms)	Controls	30	2.44±0.57	0.99(NS)
	Cases	30	2.44 ±0.52	
Amplitude (mV)	Controls	30	13.34±8.19	0.18(NS)
	Cases	30	18.28±18.52	
NCV (m/s)	Controls	30	56.10±13.47	< 0.001*
	Cases	30	45.23±3.97	

*** Statistically Significant**

NS-Non Significant

CHART 1



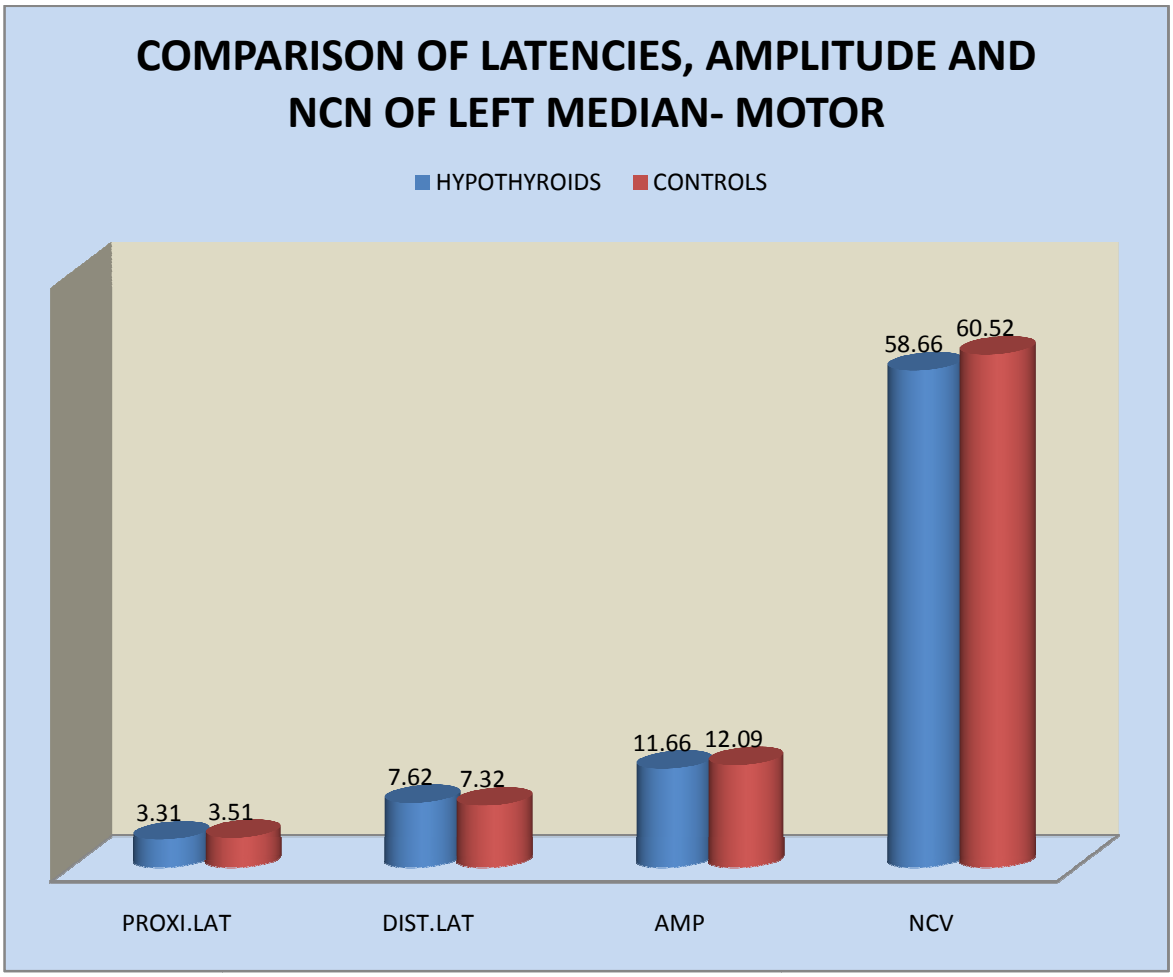
PROX.LAT: PROXIMAL LATENCY

DIST.LAT: DISTAL LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 2



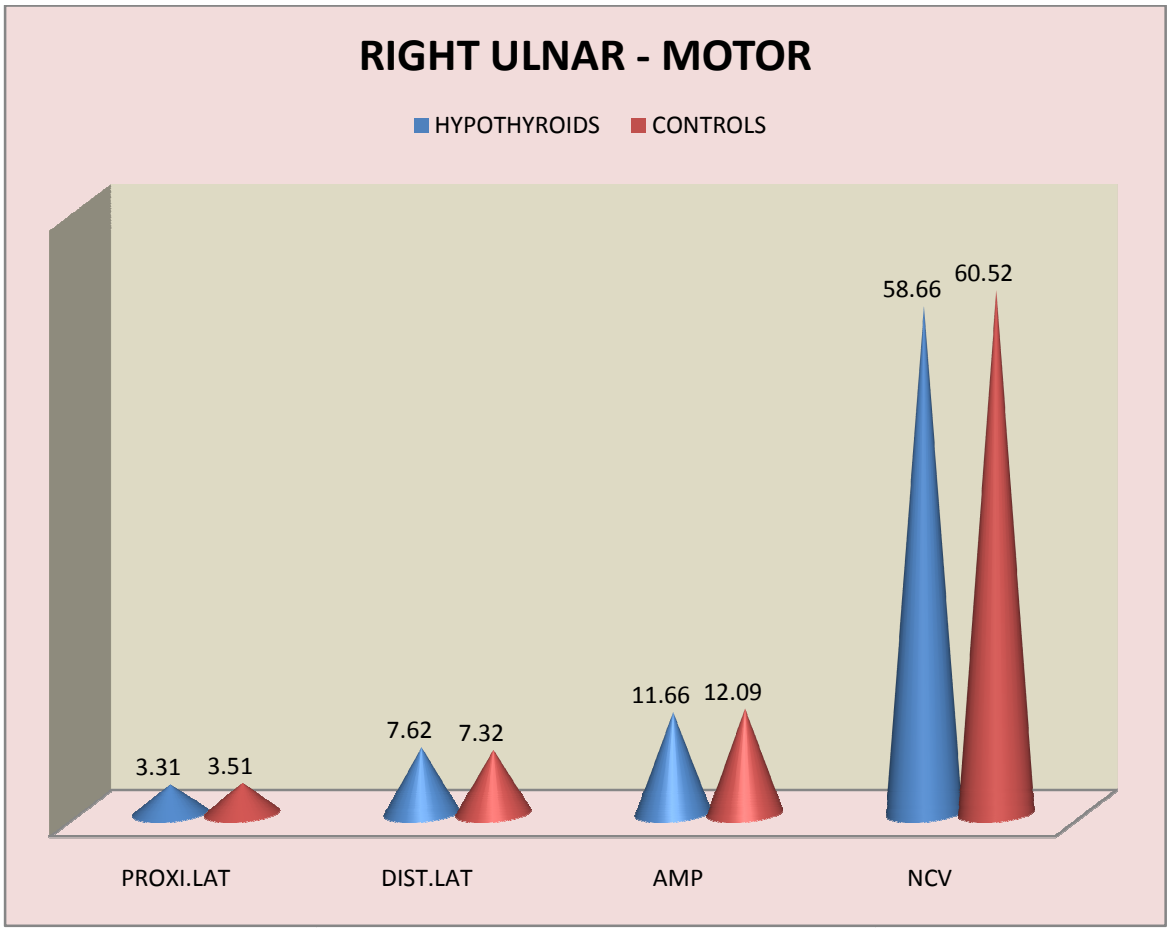
PROX.LAT: PROXIMAL LATENCY

DIST.LAT: DISTAL LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 3



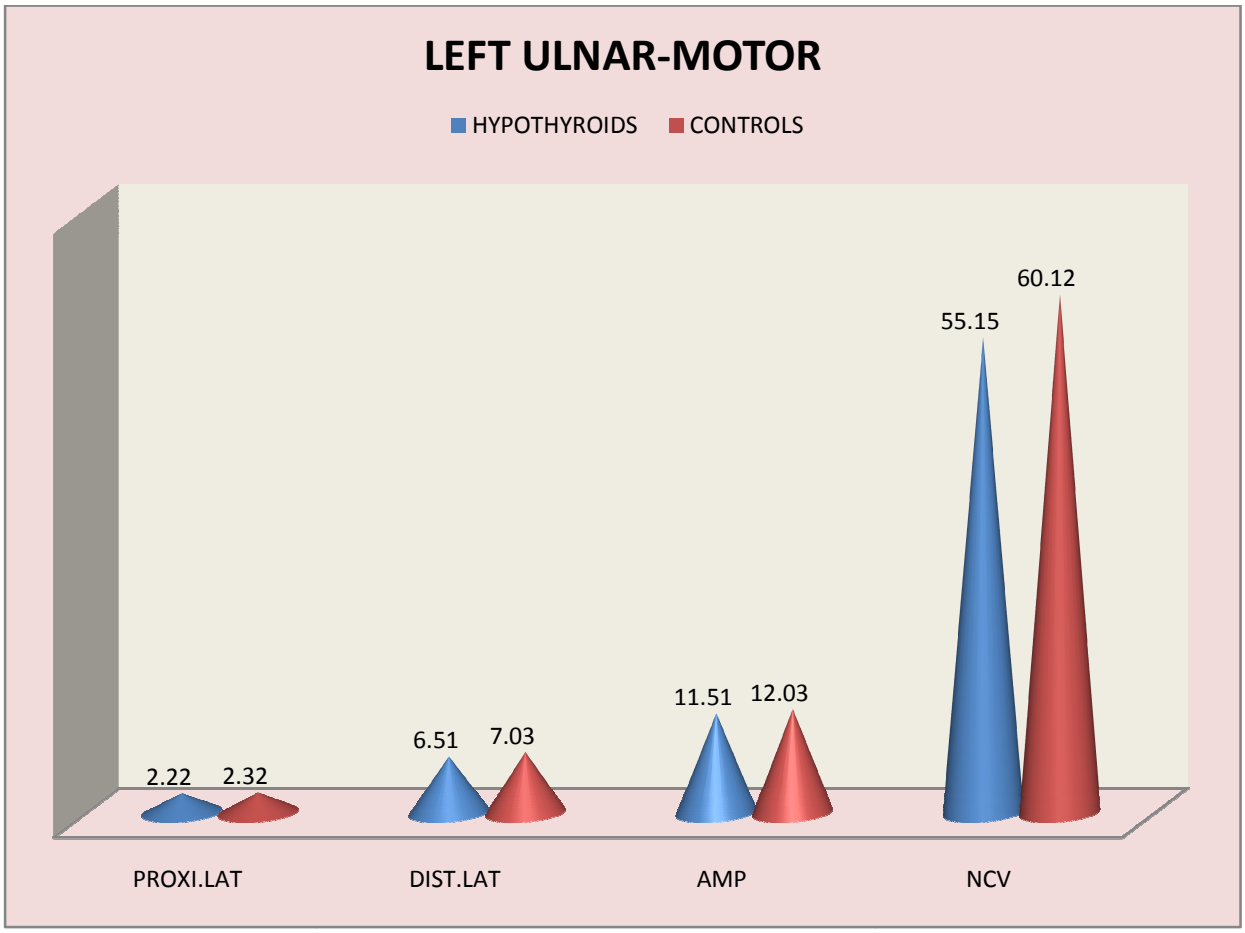
PROX.LAT: PROXIMAL LATENCY

DIST.LAT: DISTAL LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART4



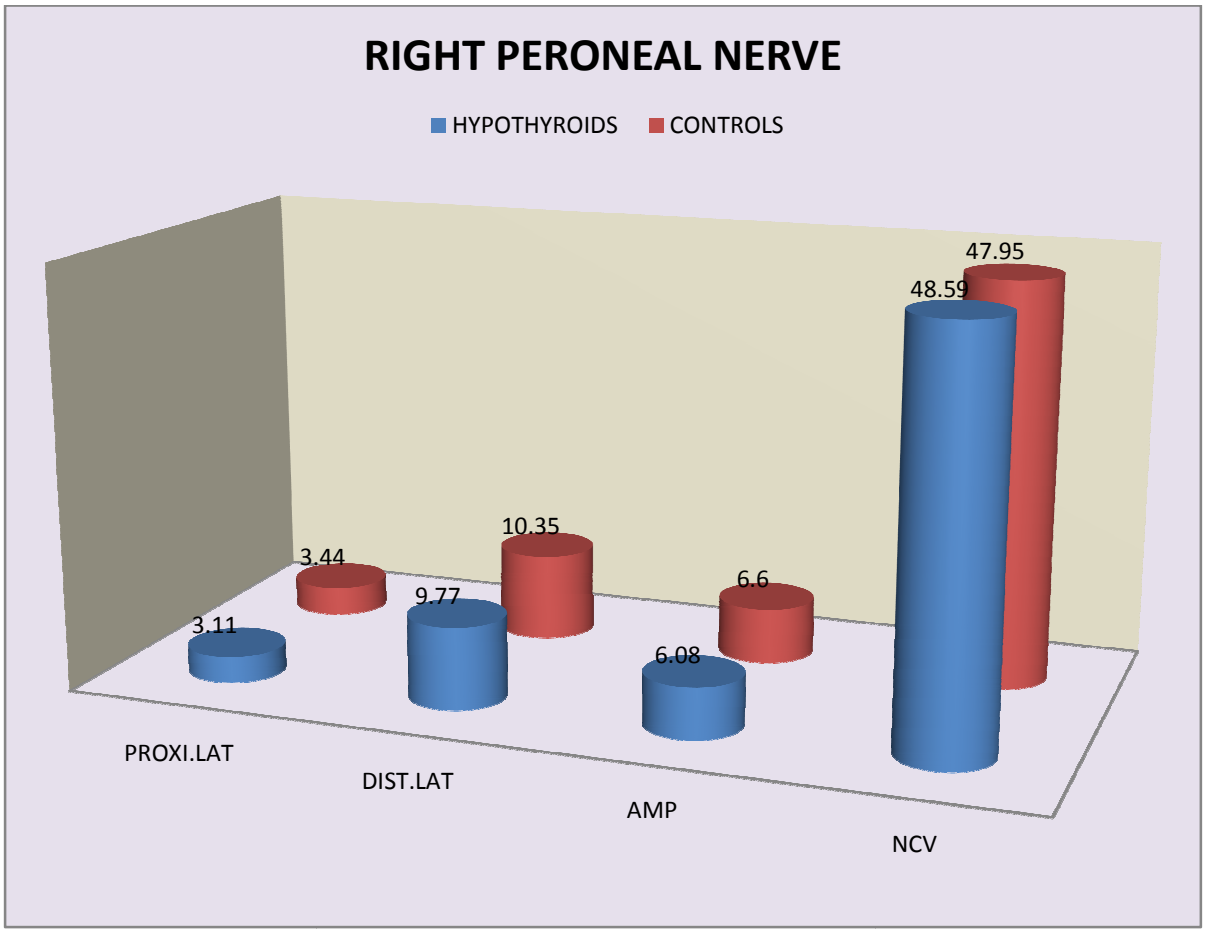
PROX.LAT: PROXIMAL LATENCY

DIST.LAT: DISTAL LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 5



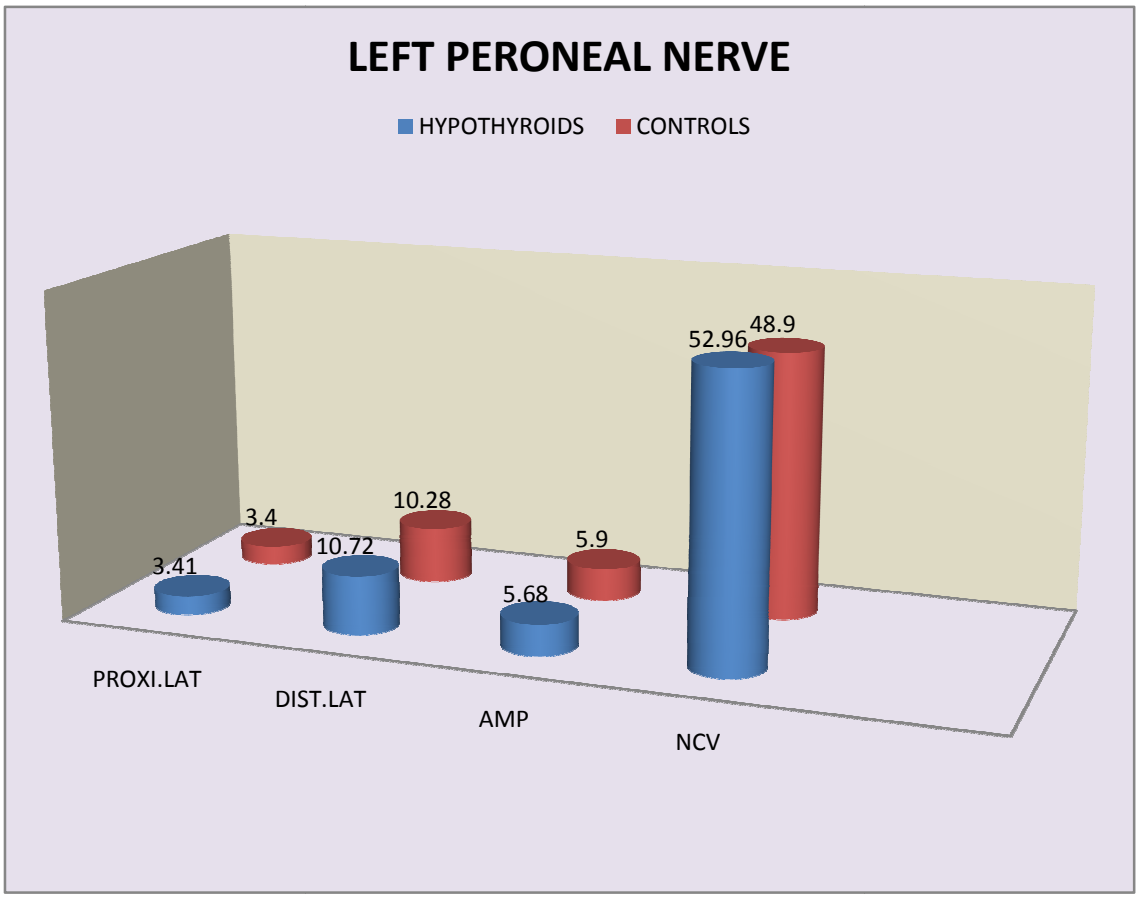
PROX.LAT: PROXIMAL LATENCY

DIST.LAT: DISTAL LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 6



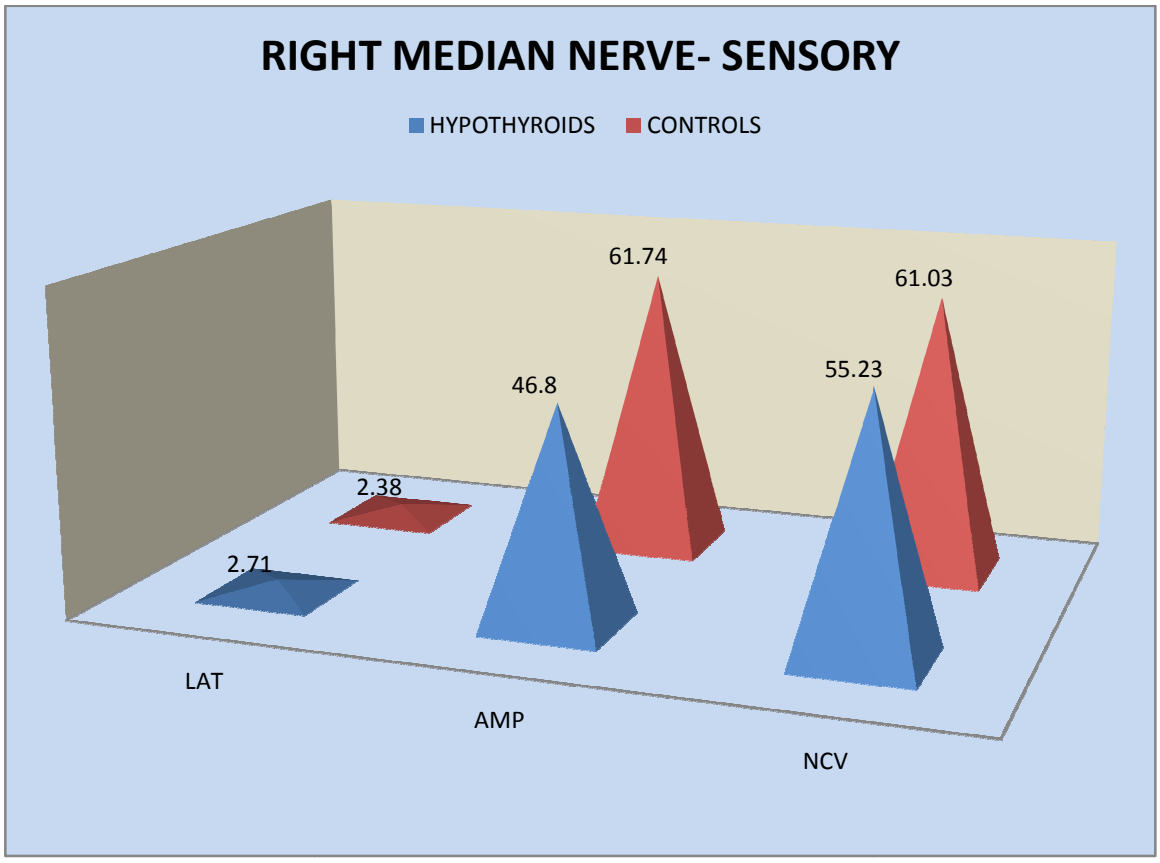
PROX.LAT: PROXIMAL LATENCY

DIST.LAT: DISTAL LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 7

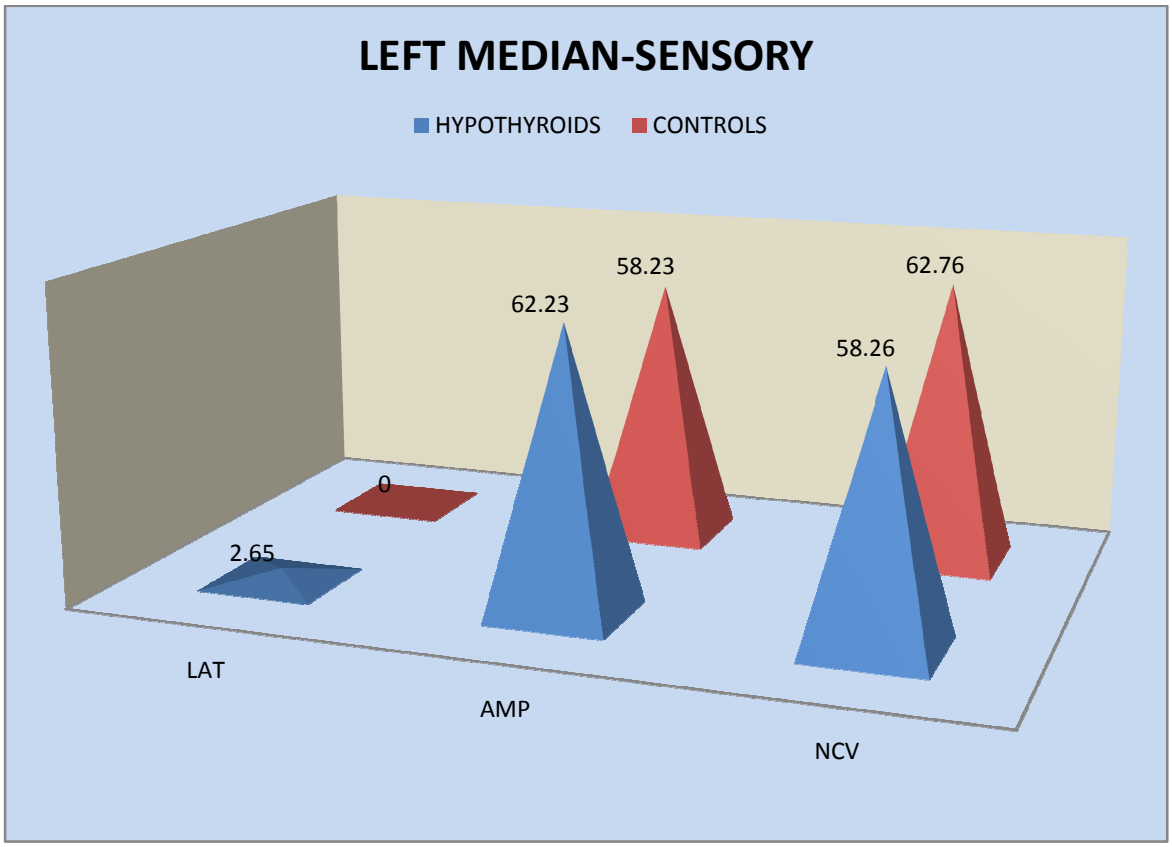


LAT: LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 8

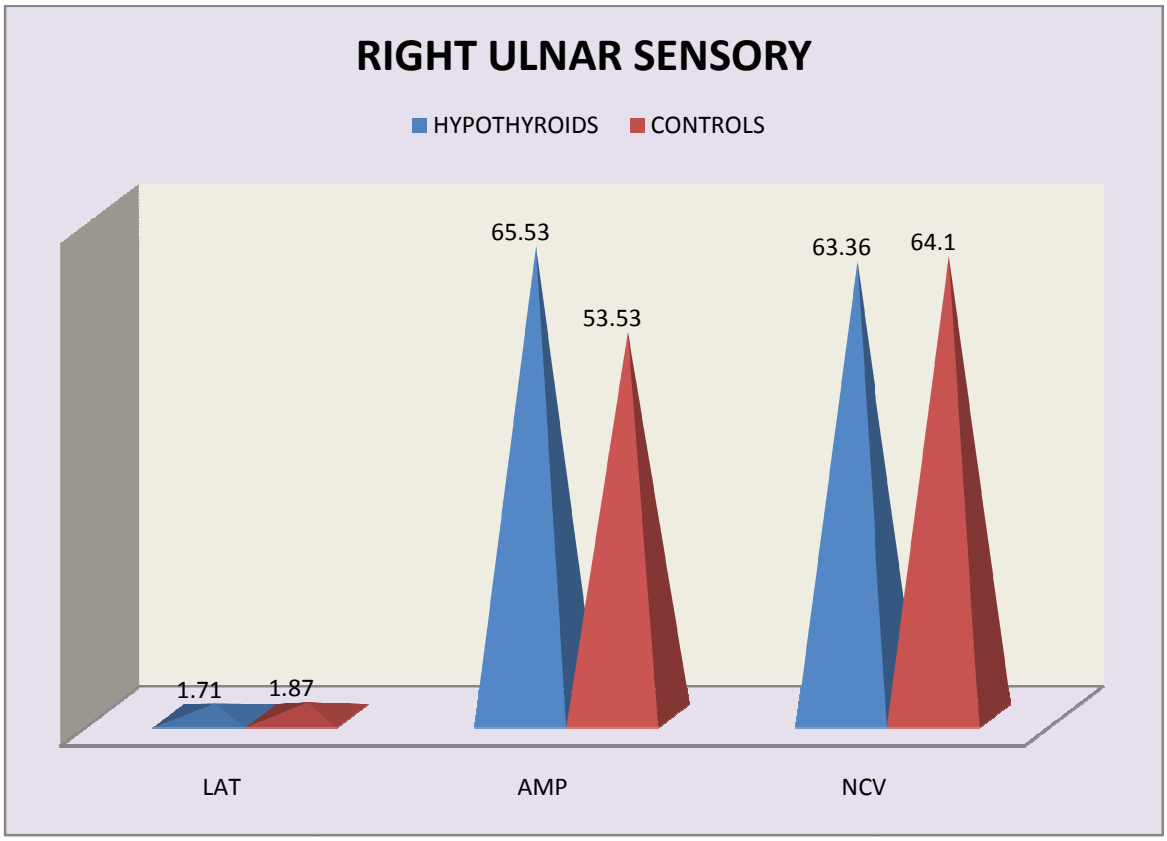


LAT: LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 9

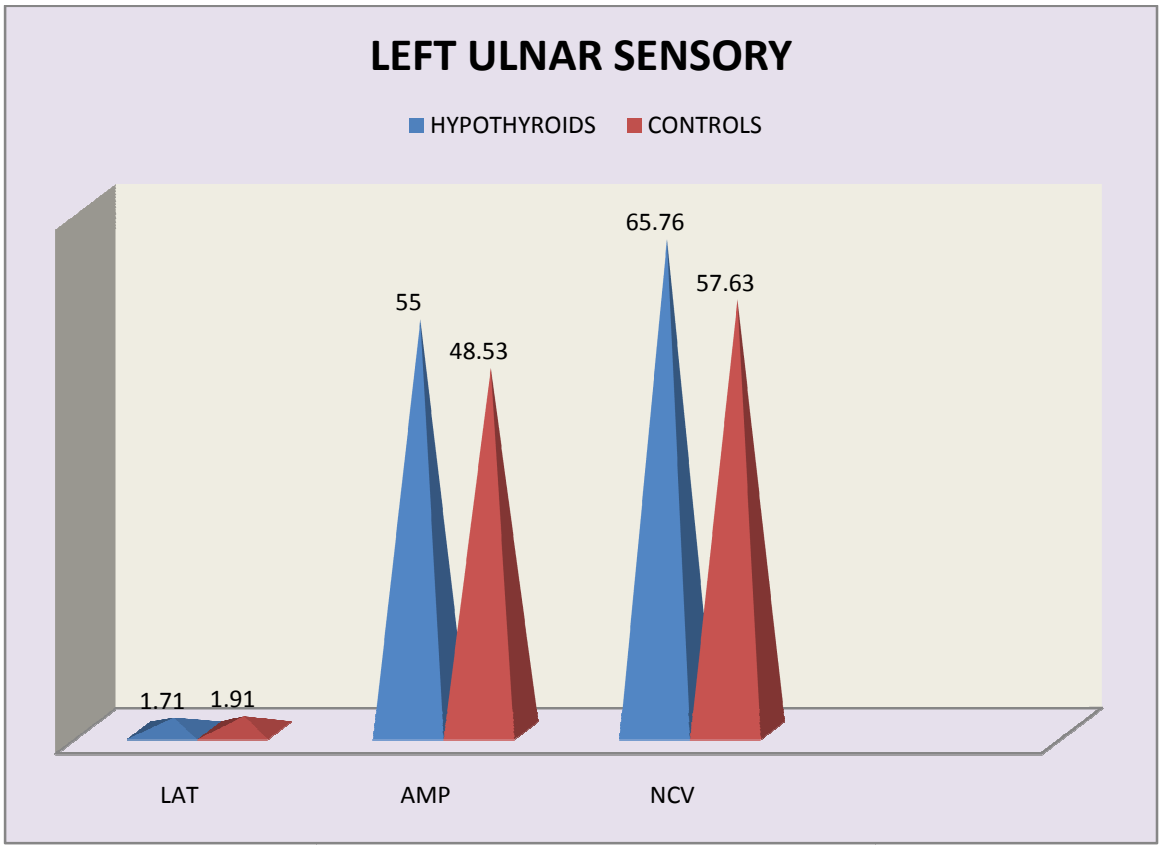


LAT: LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 10

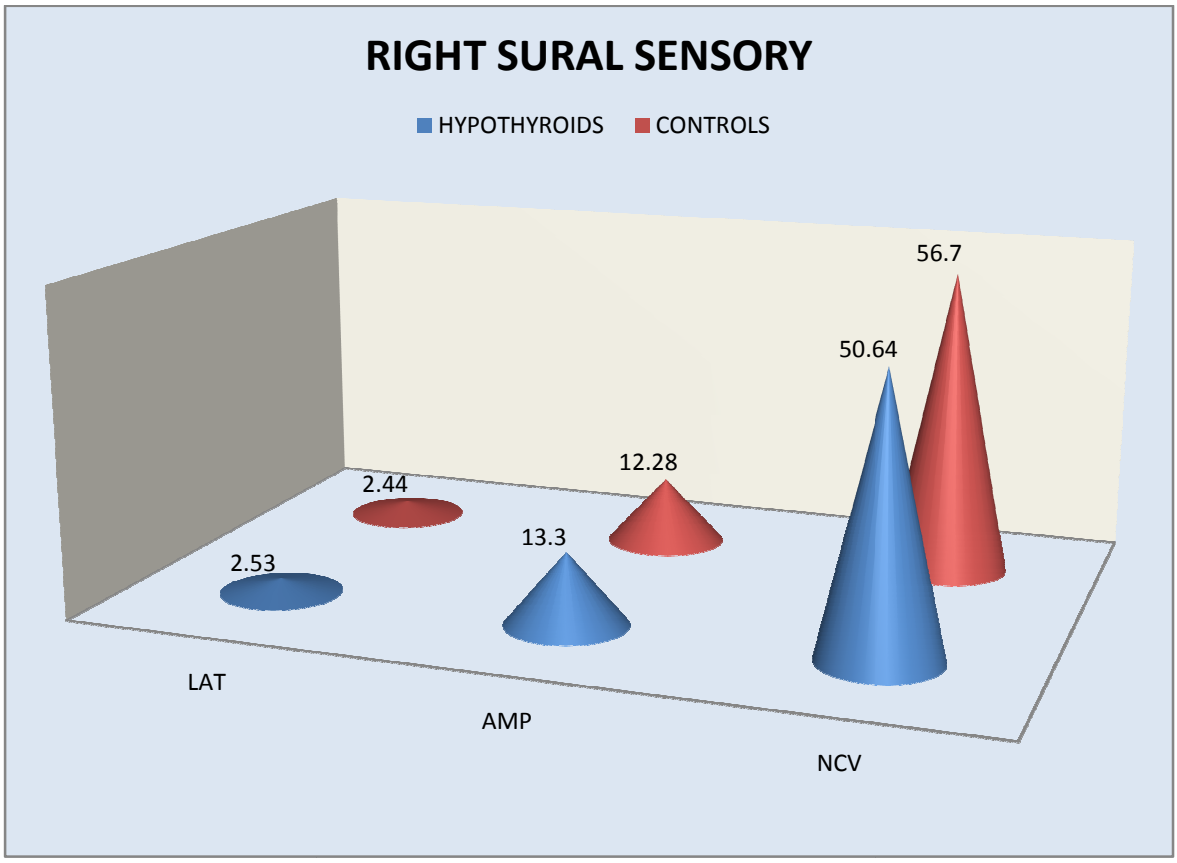


LAT: LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 11

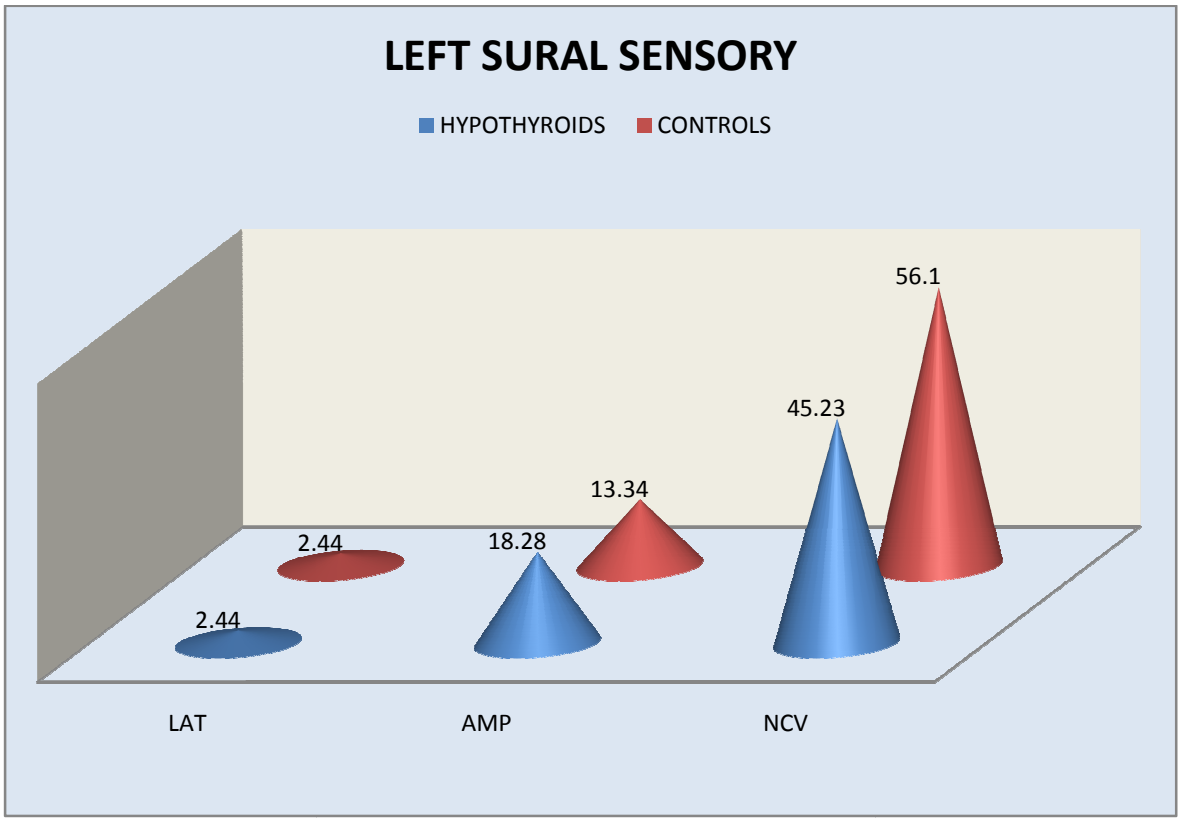


LAT: LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 12

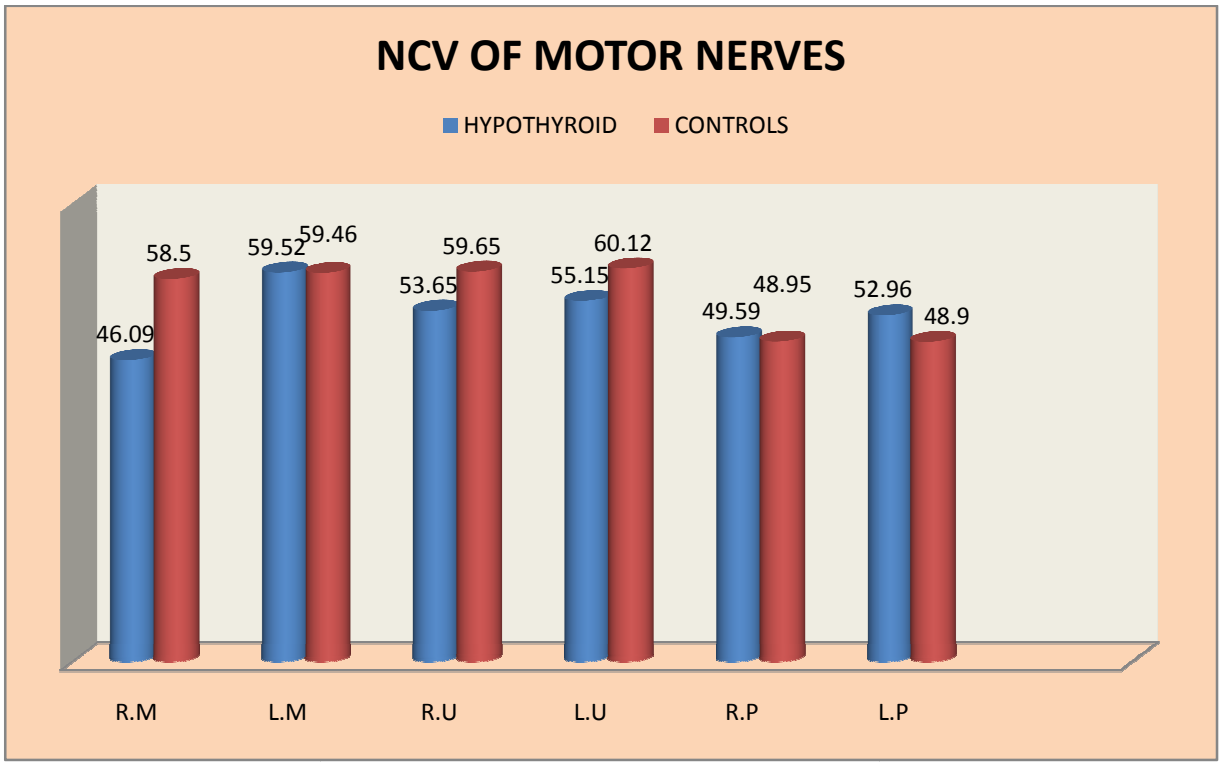


LAT: LATENCY

AMP: AMPLITUDE

NCV: NERVE CONDUCTION VELOCITY

CHART 13



NCV= NERVE CONDUCTION VELOCITY

R.M= RIGHT MEDIAN NERVE

L.M=LEFT MEDIAN NERVE

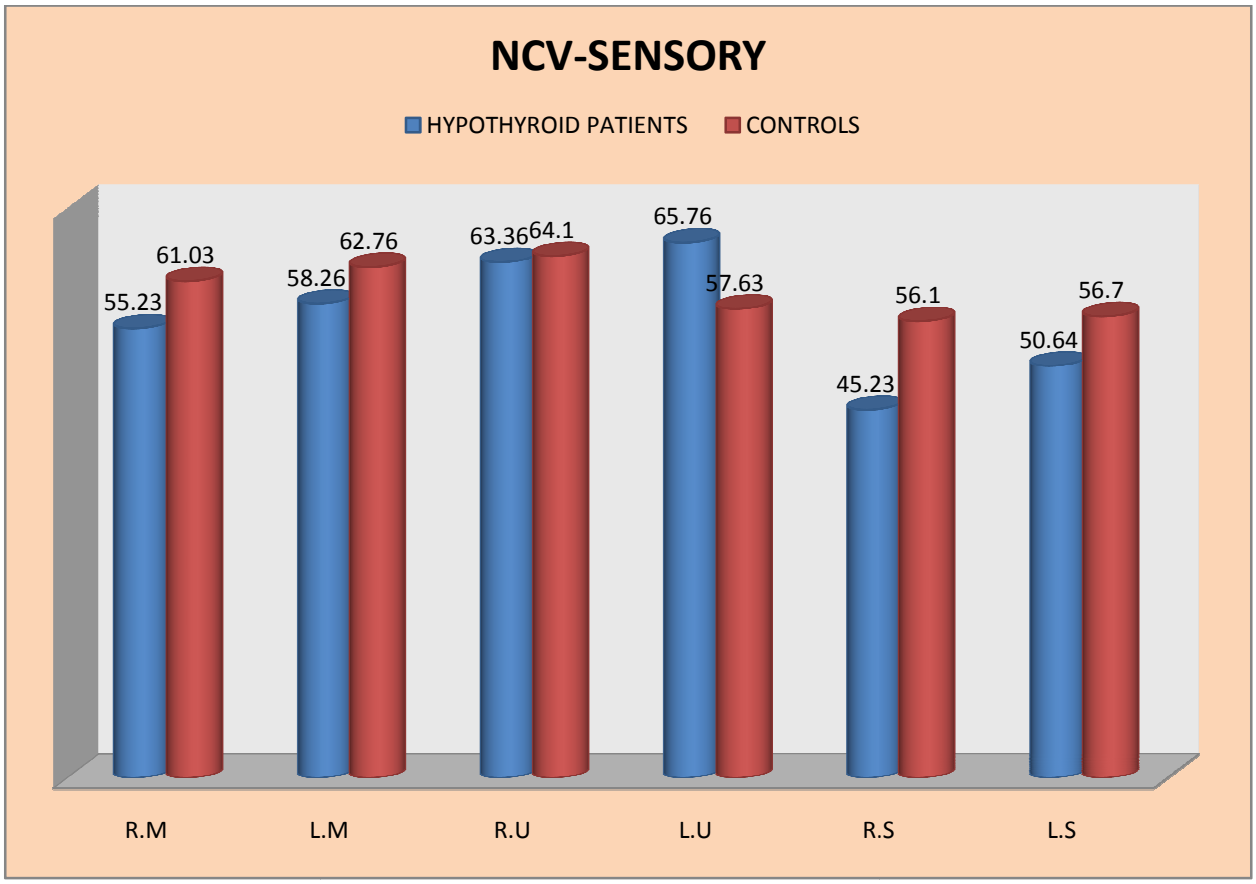
R.U=RIGHT ULNAR NERVE

L.U= LEFTULNAR NERVE

R.S= RIGHT PERONEAL NERVE

L.S= LEFT PERONEAL NERVE

CHART 14



NCV= NERVE CONDUCTION VELOCITY

R.M= RIGHT MEDIAN NERVE

L.M=LEFT MEDIAN NERVE

R.U=RIGHT ULNAR NERVE

L.U= LEFTULNAR NERVE

R.S= RIGHT PERONEAL NERVE

L.S= LEFT PERONEAL NERVE

DISCUSSION

DISCUSSION

Hypothyroidism is an endocrine disorder of deficient thyroid hormone levels in the circulation. It can affect multiple system in our body including nervous system, musculoskeletal system, cardiovascular system, respiratory system, gastrointestinal system, reproductive system and genitourinary system.Thyroid hormones are essential for the normal functioning of the brain and nervous system[8].

Endocrine manifestations depend on the cause of the disease, duration and severity of hypothyroidism. Nerve conduction study (NCS) provides the greatest help in assessing the peripheral nerve disorder. Prolonged nerve conduction time, decreased amplitude and longer latencies are the well documented features of neurological findings in hypothyroidism and that can be reversed with treatment[31]. More common presenting feature is the carpal tunnel syndrome with 29% incidence[13]. The connective tissues of the tendon get thickened and entrap the median nerve, which is the reason for the carpal tunnel syndrome.

In our present study we find that the nerve conduction velocity is reduced in right median, right and left ulnar and left common peroneal nerves. Somey G et al [26] reported slowed nerve conduction velocity in median, ulnar and sural nerves.

The latencyis prolonged in the right and left ulnar nerve as well as in the peroneal (both right and left) nerve in our present study. In the right ulnar nerve both the proximal and the distal latencies are prolonged, where as in left ulnar the distal latency is prolonged. The distal latency of the both side of the peroneal nerve is abnormally increased. A study done by EtorreBeghi et al[33] showed the most commonly affected parameter was the increased distal latency of the peroneal nerve with incidence of 36%.

The amplitude of the nerve conduction action potential of the all the nerves included in our study is not significantly reduced. Jane Martin[36] reported in their study that they found the sensorimotor axonal neuropathy in their patients with nerve conduction abnormalities. 60% of their patients were presented with diminished peripheral sensation and showed absent or reduced

sensory action potential, prolonged latencies and minimal decrease in nerve conduction velocity with more of axonal neuropathy than with segmental demyelination.

The following three types of pathological changes can affect the impulse conduction in a nerve[21],

1. Axonal degeneration

2. Axonal regeneration and

3. Demyelination or remyelination.

If the entire neuron is damaged or if a distal segment of the neuron is disconnected from the cell body in injury that leads to the death of the neuronal cell body. Hence the axonal degeneration occurs, which can also occur as a 'dying back' phenomenon in the most distal part of the nerve[21].

The conduction velocity is a measure of the faster conducting fibres. So unless the pathological disturbance occurs selectively in the largest myelinatedfibres, the loss will be random, that is even 75% of the axonal

population is reduced, many of the quite fast conducting fibres will be functionally active and the conduction velocity be mildly altered[39].

The sensory nerve conduction velocity of the sural nerve is reduced in our present study. Ploalapenza et al[20] also showed the decreased sensory conduction velocity of the sural nerve. Dyck and Lambert[9] studied the sural nerve biopsy by an electron microscope and demonstrated both axonal degeneration and segmental demyelination with remyelination. They stated thatthe histological findings are due to the abnormalities of the Schwann cell metabolism or axonal degeneration or remyelination.

The sensory action potentials will be of low amplitude or absent as the axonal degeneration reduces the number of the functioning nerve fibers. The number of the motor axons is diminished, which results in reduced amplitude of compound muscle action potential.

Demyelination is due to the loss of the myelin sheath of the axon, where the axon tubule is intact. The functional recovery is better in this

demyelination when compared to axonal degeneration. It may be paranodal or segmental demyelination. The former will block the conduction where as the later can only reduce the conduction velocity[21].

In a study done by Gulbun Yuksel[5], the most affected nerve median (54%) motor and sensory nerves followed by the sural nerve (18%). This study supports our finding that in our study also the most affected nerve is median nerve. In this present study 16 patients found to have carpal tunnel syndrome (53.33%) among them tenpatients (33.33%) had bilateral carpal tunnel syndrome. Six patients (20%) had median nerve sensory nerve abnormality. Cruz et al[32] studied the electroneuromyography and neuromuscular findings in 16 primary hypothyroid patients among them 43.7% had CTS. In our present study sural nerve was affected in five patients out of the thirty hypothyroid patients (16%).

In this present study we could not find any correlation between the nerve conduction abnormalities and the age and duration of the disease in hypothyroid patients. Eslamian F et al[18] studied the electrophysiological changes in patients with untreated hypothyroidism and

stated that they could not find either any significant relationship between age, duration of disease, serum TSH level or the presence of neuropathy or myopathy.

Based on the duration of the hypothyroidism, we categorized our patients in to three groups as follows:

Group I: Newly diagnosed hypothyroid patients (4), that is on that visit in our hospital, when they are found to be hypothyroid.

Group II: the patients with duration of disease less than 5 years (16) and

Group III: patients with duration of disease more than 5 years (10).

In our study there is no statistically significant difference in nerve conduction values between the three groups of duration. But we found that two out of four newly diagnosed hypothyroid patients have carpal tunnel syndrome either bilaterally or unilaterally. Age and duration of the disease do not correlate with the changes in the nerve conduction parameters.

By this nerve conduction study it is found that 36.66% of hypothyroid patients have neuropathy.

On analysis of the nerve conduction study values of the three (median, ulnar and sural nerve) nerves on patients and controls the findings are follows:

a) 36.66% of hypothyroid patients show electrophysiological changes suggestive of neuropathy
b) 20% of the patients (6) show sensory abnormality in the median nerve conduction, 16.66% patients (5) show abnormal in the sural nerve .so the upper limb is more affected than the lower limb considering the sensorycomponent, according to our study.
c) Considering the parameters most affected, the latency is the most affected parameter (18%), next is the amplitude (3%). The nerve conduction velocity is reduced in patients (35.33%).

Amplitude measures are important in sensory nerve conduction evaluations.[21.] The nerve conduction velocity in turn is depend mainly on the faster conducting nerve fibre, even if maximum number of nerve fibres get affected, the presence of few faster conducting fibre carry the conduction and the result will be disproportionate to the affected fibres[21].

Both latency and conduction velocity depend on the intact, myelinated nerve fibre as the myelin and node are essential for the fast action potential propagation. In contrast, the amplitude of the wave form depends primarily on the number of the axons functioning within the nerve. Slowing conduction velocity or prolongation of latency usually implies demyelinating injury, while loss of amplitude usually correlates with axonal loss or dysfunction[39].

In this study electromyography was not done. Our study did not include the autonomic function test and all other peripheral nerves. These are the limitations in our study

.

CONCLUSION

CONCLUSION

The prevalence of neuropathyis 56.66 % among the hypothyroid patients attending the PSG Institute of Medical Science and Research, Coimbatore. 36.66% of the hypothyroid patients (11) were found to be with carpal tunnel syndrome.

The physiological parameters (age and duration of the disease) are not correlated with nerve conduction values.

The median nerve is the most affected nerve in the upper limb and the sural nerve is the commonly affected nerve in the lower limb.

Estimation of the nerves conduction values can be considered as a useful parameter in the diagnosis and evaluation of the neuropathy in hypothyroid patients. The presence of carpal tunnel syndrome without clinical neuropathy, suggests that nerve conduction study can be carried out as a routine investigation to find out the electrophysiological alterations without clinical presentation in hypothyroid patients.

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ANNEXURE

PROFORMA

PATIENT DATA:

1. **Patient Name:**
2. **IP/OP Number:**
3. **Sex:**
4. **Age (Yrs):**
5. **Height (Cm):**
6. **Weight (Kg):**
7. **Occupation:**

II. PERSONAL INFROMATION

Address :

City :

Phone No :

Mobile No :

III. HISTORY

1. **Complaints if any:**
2. **Past History:**

3. **Personal History:**

4. **Smoking :**

5. **Alcohol :**

6. **Hypertension :**

7. **Other Illness :**

(Peripheral Neuropathy , Arthralgia or Others)

8. **Family History of Hypothyroidism:**

HYPOTHYROIDISM HISTORY

9. **Duration (in years) :**

10. **Treatment :**

11. **Regularity :**

GENERAL EXAMINATION

1. **Pulse /min :**
2. **BP :**
3. **Anemia :**

SYSTEMIC EXAMINATION

1. **CVS :**
2. **RS :**
3. **ABDOMEN :**

NEUROLOGICAL EXAMINATION

1. **HISTORY**

Symptoms of neuropathy, if present or not

a. **Tingling / Hyperesthesia**

b. **Decreased sensation**

c. **Numbness**

2. EXAMINATION OF NERVOUS SYSTEM

A. SENSORY SYSTEM

a. **Touch**

b. **Pain**

c. **Position sense**

B. MOTOR SYSTEM

a. **Nutrition**

b. **Power**

c. **Reflexes**

INFORMED CONSENT

I Mr / Mrs. _____ age _____ have been adequately and well explained in the language that I understand with my full consciousness regarding the nerve conduction tests, that to do done on me. The purpose of this study, the necessity of tests, the little discomfort while stimulating that nerves were all explained to me prior to the tests being done in detail.

After understanding the basis of this study, I give my full consent to the study with utmost normal mental health.

Truly,

_____

(Signature of the subject)

Name-

Date & Time

(To be filled by the subject)

RAW DATA

RIGHT MEDIAN MOTOR (CASES)						RIGHT MEDIAN MOTOR (CONTROL)				
S.NO	A1 Lat.ms	Amplitude	A2	Ampli	CV	A1 Lat.ms	Amplitude	A2	Ampli	CV
1.	3	14.7	6.2	14.3	78	3.2	9.9	7.6	9.2	55
2.	4.7	14.4	8.5	14	66	3.8	13.3	8.1	12.7	53
3.	4	0.1	7.5	0.1	71	3.7	10.6	7.6	8.9	64
4.	4	14.3	8.3	12.6	60	3.7	11.8	8.6	11.8	51
5.	3.8	10.4	7.9	9.9	61	3.4	13.6	8.3	13.9	51
6.	2.2	14.2	5.8	14.8	69	3.6	7.8	8.6	7.7	50
7.	2.3	16	6.2	17.5	64	3.5	8.5	7.6	8.8	61
8.	4.1	9.7	7.3	8.9	69	2.8	15.4	7.7	15.1	51
9.	2.8	7.6	6.98	7	59.95	3.7	11.3	8	11.9	65
10.	2.9	18.2	6.9	13.3	60	2.8	13.7	6.6	13.4	66
11.	3.3	11.2	7.4	10.8	61	4	11.9	8.2	11.8	51
12.	3.65	11.4	8.23	10.1	54.59	2.9	11.9	7.2	11.9	60
13.	4.9	9.6	9.2	10.7	60	3.4	8.2	7.7	8.2	56
14.	5.8	9.9	11	9.7	60	4.4	9.3	9.3	7.8	55
15.	3.9	6.6	7.2	6.7	73	3.4	9.9	7.9	8	56
16.	2.6	10.8	6.1	10.4	71	3.3	7.1	6.9	7.3	67
17.	3.4	6.6	7	6.3	67	2.8	12.8	7	13	60
18.	2.9	14.8	6.8	15.9	62	3.7	8.4	8	7.7	63
19.	3	8.8	6.6	8.5	67	3.3	16.1	7.7	15.3	55
20.	3.1	15.9	7.2	15.7	61	3.3	16.1	7.7	15.3	55
21.	3.6	12.4	8	12	55	3.7	8.2	7.7	8.2	60
22.	2.6	16.6	6.3	14.9	62	3.4	11.3	7.4	10.5	65
23.	2.7	13.6	6.4	14.6	65	3.4	11.3	7.4	10.5	65
24.	4	13.8	7.8	14.6	63	3.4	11.3	7.6	10.5	62
25.	5.7	9.5	10.1	8.8	57	3.4	18.4	8.2	18.5	58
26.	5.6	9.1	4.5	9.9	27	3.1	19.4	7.1	18.8	63
27.	3.5	15.5	7.6	14.9	63	3.7	8.2	7.7	8.2	60
28.	3.4	13.3	8	13.4	54	3.3	9.9	7.9	8	56
29.	4.2	9.9	8.2	8.6	56	4.3	9.2	9	8.1	56
30.	4.3	13.3	8	13.4	54	3.3	15	7.8	15.2	55

LEFT MEDIAN MOTOR (CASES)						LEFT MEDIAN MOTOR (CONTROL)				
S.NO	A1 - Lat.ms	AMPLI	A2	AMLI	CV	A1 - Lat.ms	AMPLI	A2	AMLI	CV
1.	3.2	12.7	6.5	9.6	76	2.4	15.1	6.8	15.9	57
2.	3.8	12.1	7.4	11.2	69	3.3	13	7.5	11.9	61
3.	6.2	0.3	3.1	0.4	81	3.5	13.3	7.6	11.8	61
4.	5.1	11.4	9.3	10.5	60	3.9	11.8	8.6	11.8	53
5.	3.7	7.7	7.9	12.1	60	3.2	14.6	7.8	12.4	54
6.	2.2	13.5	6.2	14.2	63	3.4	11.2	8.2	11	50
7.	2.3	16.4	6.2	18.2	64	3.3	9.6	7.4	9	61
8.	3.6	9.5	7.3	8.7	68	3.4	12.9	8	12.8	54
9.	2.29	11.5	6.67	10	57.08	3.4	13.7	7.8	12.9	64
10.	3.3	13.6	7.3	15.1	63	2.6	10.8	6.9	8.9	58
11.	2.8	14.6	6.7	13.5	64	3.9	11.9	8.2	12.1	54
12.	2.71	18.2	6.98	17.7	58.55	3.5	10.9	7.3	8.2	68
13.	3.4	17	7.6	17	57	3.2	8.2	7.3	8.4	59
14.	6.6	9.9	11	9.7	57	4	9.3	8.4	7.7	57
15.	2.9	16.7	7.2	11.9	53	3.2	9.8	7.7	8.2	56
16.	2.2	12.2	6.1	10.4	64	3.4	7.4	7.5	6.7	59
17.	3	12.8	6.8	11.7	63	3	13.6	7	12.7	63
18.	2.6	15	6.8	14.9	57	3.4	10.5	8.3	10.5	51
19.	3.3	8.6	7.3	9.3	60	3.4	13	7.2	13.2	66
20.	3.1	14.9	6.9	15.6	66	3.4	13	7.2	13.2	66
21.	3.2	12.4	8	11.5	50	3.4	8.2	7.3	8.4	62
22.	2.6	16.5	5.8	14.9	72	2.9	17.5	7.4	14.6	58
23.	2.6	13.6	6.4	14.6	63	2.9	17.4	7.4	14.4	58
24.	3.8	13.8	7.8	14.5	63	3.2	17.5	7.5	14.6	60
25.	5.4	9.5	9.5	8.8	61	3.4	20.1	8.3	19.9	51
26.	5.1	9.5	9.8	8.8	53	3.5	15.5	7.6	14.9	63
27.	3.1	19.4	7.1	18.8	63	3.4	8.2	7.3	8.4	62
28.	3.9	14.4	7.9	12.4	63	3.2	9.8	7.8	8.1	55
29.	3.4	9.5	8.2	4.5	52	3.5	10	7.2	8.3	67
30.	3.9	14.4	7.9	12.4	63	3.3	19	8.1	19	52

RIGHT ULNAR MOTOR (CASES)						RIGHT ULNAR MOTOR (CONTROL)				
S.NO	A1 Lat	Ampli	A2 Lat	Ampli	CV	A1 lat	Ampli	A2 Lat	Ampli	CV
1.	1.9	11.1	6.5	12.6	57	1.88	9.2	6.46	9.4	56.77
2.	1.9	17	5.4	16.4	74	1.98	14.6	6.46	13.7	58.04
3.	2.6	15.6	7.9	14.7	51	1.8	14.4	6.3	14.3	58
4.	2.4	8.7	6.9	7.6	59	2.5	10.4	7.9	9.4	50
5.	2.92	8.9	5.94	7.8	43.77	2.3	12	6.9	11.9	57
6.	2.4	15.7	7.6	14.6	50	2.5	16.5	7.6	15.2	51
7.	1.9	13.5	6.1	11.1	64	2.3	17.8	7.6	16.9	51
8.	2.1	22.7	6.3	22	62	2.5	9.1	7.8	8.9	51
9.	2.5	12.1	7.8	12.1	51	2.6	11.6	7.5	11.3	51
10.	2.5	9.1	6.3	8.9	66	2.3	10.5	7.2	12.4	53
11.	1.9	9.2	6.3	7.9	59	1.9	11.2	7.5	11.4	50
12.	2.2	14.9	5.8	13.4	69	2.7	14	8.2	13.3	55
13.	2.1	14.6	5.7	13.4	72	2.3	16	8	13	52
14.	2.2	11	6.2	11.5	63	2	15.9	7	15.9	52
15.	2.2	13.8	6.9	14	55	2.5	12.2	7.4	9.5	53
16.	2.1	10.5	6.2	8.6	63	2.5	8	6.1	7.6	69
17.	1.7	13.6	6.7	12.1	52	2.4	11.3	7.6	13	52
18.	2.2	15	6.6	15.2	64	2.7	13.2	8.2	11.4	51
19.	2.2	15	6.6	15.2	64	1.9	8.6	6.5	7.2	54
20.	2.3	12	7	10.5	55	2.4	14.1	6.9	15.2	58
21.	2.1	15.8	6.7	14.4	57	2.1	18	7.3	14.7	51
22.	2.9	12.2	7.4	9.5	58	2.6	9.6	7.8	8.8	50
23.	2.1	15.8	6.7	14.4	57	2.6	10.6	7.7	8.9	51
24.	1.8	7.1	6.3	6.3	62	2.5	11.4	8	11	50
25.	2.5	14	6.5	11.7	65	2.5	11.2	8.1	11.3	51
26.	2.9	19.2	6.6	19.1	70	2.3	11.9	8.6	11.5	53
27.	2	13.8	6.9	12.4	53	2.6	11.6	7.5	11.3	51
28.	1.6	13.2	6.6	12	50	2.3	10.5	7.2	12.4	53
29.	1.9	12.6	5.9	12.4	63	1.9	11.2	7.5	11.4	50
30.	1.25	18.5	6.67	18.2	61	2.7	14	8.2	13.3	55

LEFT ULNAR MOTOR (CASES)						LEFT ULNAR MOTOR (CONTROL)				
S.NO	A1 Lat	Ampli	A2 Lat	Ampli	CV	A1 Lat	Ampli	A2 Lat	Ampli	CV
1.	1.8	7.7	5.8	8.3	65	1.25	11.2	6.25	9.7	52
2.	2.1	8.1	6.2	8.5	63	1.67	14.1	6.15	13.5	58
3.	2.7	10.4	7.5	10.1	54	1.29	14	6.1	13	58.4
4.	2.2	15	6.7	14.9	59	2.3	12.1	7.5	11.7	50
5.	2.71	6.6	6.68	5.5	37.79	2.8	12.9	6.6	11.7	66
6.	2.2	10.8	6.6	10.4	61	2.2	16.5	7.3	15.2	51
7.	2.4	17.9	6.4	12.5	65	2.6	18.7	7.6	16.5	52
8.	2.2	13.3	6.5	13	62	2.3	11.2	7.5	11.7	52
9.	2.3	10.6	7.5	8.8	52	2.4	12	7.5	11.1	50
10.	2	13.9	5.7	12.8	68	2.4	13.5	7.5	11.3	51
11.	2	11	6.4	9.1	59	2.4	13	7.9	13.3	51
12.	1.8	15.3	5.8	14	63	2.4	15.5	7.4	13.3	60
13.	2.2	14.6	5.7	13.4	74	2	11.5	5.8	9.9	68
14.	2.6	11	6.5	11.5	64	2.1	17.3	6.8	16.5	55
15.	2.2	13.8	6.4	13.7	62	2.6	11.8	7.8	9.3	50
16.	2.5	10.7	6.5	8.7	65	2.5	7.9	6.1	7.6	68
17.	1.8	14	6.7	12	53	2.3	11.8	7.3	10.4	54
18.	2.3	15.1	6.7	15.2	59	2.7	14.7	8.2	14.5	51
19.	2.3	15.1	6.7	15.2	59	1.9	8.3	6.5	7.4	53
20.	2.6	11.7	7	10.5	59	2	14.8	6.3	15	60
21.	2.1	14.3	6.7	13.5	57	2	14.2	7	15.1	53
22.	2.2	10.1	6.6	8.6	59	2.8	9.8	7.8	9.3	52
23.	2.1	14.3	6.7	13.5	57	2.8	9.9	7.6	9	53
24.	2.1	7.1	6.6	6.3	62	2.1	11.7	7.6	11.5	51
25.	2.4	10.1	6	9.1	72	2.1	11	7	11.3	52
26.	3	19.2	7	19.1	65	2.4	12	7.5	11.1	50
27.	2.1	14.4	6.2	10.7	63	2.4	13.5	7.5	11.3	51
28.	2	13.5	6.7	12.1	53	2.4	13	7.9	13.3	51
29.	1.9	12.6	5.9	12.4	63	2.4	15.5	7.4	13.3	60
30.	1.9	12.1	7.2	11.9	49	2.7	14.7	8.2	14.5	51

RIGHT PERONEAL (CASES)						RIGHT PERONEAL (CONTROL)				
S.NO	A1 Lat	Ampli	A2 Lat	Ampli	CV	A1 Lat	Ampli	A2 Lat	Ampli	CV
1.	4.1	3.5	10	2.8	56	3.5	9.2	9.9	6.2	52
2.	4.2	6.7	11.9	4.3	43	3.5	9.2	9.9	6.2	52
3.	3.4	4.5	7.8	4.5	75	3.4	4.8	10.7	3.8	46
4.	3.6	6.3	9.9	5.4	51	3.5	4.8	10.7	4	47
5.	3.6	6.5	10	5.5	52	3.9	4.6	11.9	4.2	40
6.	3.7	4.8	10.2	4.6	51	2.3	10.4	9.3	9.7	47
7.	3.5	2.8	11.3	2	42	2.9	8.2	10.2	6.4	45
8.	3.6	4.5	10.2	4	54	3.2	9.7	11.2	6.9	41
9.	3	3.6	10.2	3	42	3.4	7.7	10.2	6	49
10.	3.1	8.1	7.8	7.7	65	3.5	3.9	9.6	3.5	54
11.	4.2	4.2	10.1	4	56	3.6	4.5	10.3	4	54
12.	2.8	8.3	9.4	7.5	50	3.4	8.5	9.8	7.6	52
13.	3	6.3	7.9	5.5	65	2.5	6.7	8.5	5.1	55
14.	3.4	6.1	9.5	5.2	52	3.9	11	12	8.9	43
15.	3	7.7	9.8	6.8	49	3.4	8.6	10.8	7.6	45
16.	3.3	7.8	10	6.5	50	3.9	4.9	11	4.6	46
17.	3.8	9.4	9.1	7.6	62	3.8	6	10.5	5	46
18.	3.6	9	9.9	8.9	52	2.9	6.1	10.3	5.4	45
19.	2.9	11.1	9.3	9.7	52	3.8	11	11.2	9.7	45
20.	3.54	6	11.5	4.9	43.36	3.5	5.2	10.5	4	47
21.	3	5	10	3.2	47	3.8	9.4	10.2	9.5	48
22.	3.7	7.1	9.9	7.2	60	4.9	6.5	11.4	6.3	51
23.	3.4	4.4	8.9	5.1	58	2.8	5.2	10	4.8	61
24.	3.4	6.1	9.5	5.2	52	2.6	5.2	8.75	4.8	53.66
25.	4.3	7.8	10.8	7.5	49	2.4	4.7	9.58	4.4	44.57
26.	3.4	4.5	7.8	4.5	75	3.5	9.2	9.9	6.2	52
27.	3.6	6.3	9.9	5.4	51	3.5	6.6	10.8	4.2	47
28.	3.6	6.5	10	5.5	52	3.7	4.8	10.2	4.6	51
29.	3.3	7.8	10	6.5	50	3.5	3.9	9.6	3.5	54
30.	3.8	9.4	9.1	7.6	62	3.5	3.9	9.6	3.5	54

LEFT PERONEAL (CASES)						LEFT PERONEAL (CONTROL)				
S.NO	A1 Lat	Ampli	A2 Lat	Ampli	CV	A1 Lat	Ampli	A2 Lat	Ampli	CV
1.	3.8	3.6	10	2.9	53	2.9	8.5	10	7.9	46
2.	4.2	6.8	11	4.5	42	2.9	8.5	10.5	8	46
3.	3.4	4.2	8.6	4.3	63	2.8	11.3	10.4	8	43
4.	3.3	5.7	9.8	5.1	51	2.8	10.4	10	8.5	44
5.	3.9	5.8	9.6	5.2	52	3.9	4	11.4	3.7	43
6.	3.5	10	10.2	9.5	49	2.6	6.1	9.1	5.6	51
7.	3.3	3.7	10.3	3	44	3.1	8	9.7	7	50
8.	4.1	4.5	10.7	4	55	3.9	10	11.2	7.7	45
9.	3.1	3.7	10.3	3.3	43	3.7	7.7	10.7	6	47
10.	3.3	8	9	2.8	54	3.6	3.6	9.9	3.1	52
11.	1.25	10	9.17	8.9	40.4	4.1	7.1	10.1	7.2	60
12.	2.8	8.5	9.7	7.6	49	3.2	8.5	10.2	7.6	47
13.	3.3	6.3	8.2	5.5	65	2.7	6.7	8.5	5.1	57
14.	2.8	5.7	9.4	5	48	3.9	10.7	11.9	9.4	44
15.	3.4	8.1	10.1	7.1	49	3.8	10	11.4	8.2	43
16.	2.7	9.5	9.8	9.4	46	3.8	6	10.7	7.7	48
17.	3.7	7.5	9.6	7	56	4.1	2.2	11.9	2.5	42
18.	3.6	9.6	10	9	53	3.5	5.6	11.4	5	42
19.	2.8	11.4	9.4	9.9	52	3.3	11	11.4	8.7	41
20.	3.65	4	11.98	3.5	39.62	3.2	10	10.2	8.4	44
21.	3.4	4.9	10	3.3	50	3.6	8	10.2	7	47
22.	3.7	7.6	9.9	7.4	52	4.9	6.5	11.4	6.3	51
23.	3.2	4.7	8.9	5.1	56	3.3	7.8	9.9	7.1	50
24.	2.8	5.7	9.4	5	48	3.44	5.4	8.75	5.1	62.15
25.	4.2	9	11.2	9.2	46	3.54	8.6	9.79	7.2	51.2
26.	3.4	4.2	8.6	4.3	63	2.9	8.5	10	7.9	46
27.	3.3	5.7	9.8	5.1	51	3.3	6.4	10.8	5.6	45
28.	3.9	5.8	9.6	5.2	52	3.5	10.2	10.2	9.5	49
29.	2.7	9.5	9.8	9.4	46	3.6	3.6	9.9	3.1	52
30.	3.7	7.5	9.6	7	56	3.6	8	10	3.5	51